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Original Articles.

ON DISTURBANCES OF SENSATION WITH ESPECIAL REFERENCE TO THE PAIN OF VISCERAL DISEASE.[1]

BY HENRY HEAD, M.A., M.D.

University College Hospital.

INTRODUCTORY.

SEVERAL years ago I was led to examine the positions occupied by pain in disorders of the stomach, and I soon came to the conclusion that the usual description was incomplete in several respects. For firstly the positions over which the patient experienced pain in gastric disturbances were more numerous than was usually supposed, and secondly the pain was in many cases associated with definite cutaneous tenderness. Moreover the cutaneous tenderness was in many cases not confined to small spots or areas, but occupied whole tracts of skin with definite borders. I was thus led to investigate the pain and accompanying tenderness consequent on disturbances of other organs, and I found that these sensory disturbances also followed definite lines.

After Ross's most suggestive papers it seemed exceedingly probable that these areas bore some definite relation to nerve-distribution, and I then began to investigate the distribution of herpes zoster in the hope that a skin lesion which was notoriously of nervous origin might throw some light on the meaning and significance of the tender areas in visceral disease. To my surprise I found that the areas occupied by herpes zoster exactly corresponded to those with which I was familiar in visceral disease.

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[1] Read as a Thesis before the University of Cambridge, June, 1892, and before the Neurological Society of London, November 10th, 1892.

I next attempted to determine to what level of the nervous system these areas belonged, with the help of cases in which gross organic lesions were present. By this means it became apparent that each of these areas represented the distribution of either a single nerve root or of a single segment in the spinal cord.

Thus I was enabled to map out the areas supplied by the various segments of the cord on the surface of the body. It then became apparent that certain of these areas were never affected in visceral disease, and this led me to examine the sensory supply of the viscera from the sympathetic system. Now Ross had already suggested that in visceral disturbances pain (and therefore in my cases tenderness) was referred along the distribution of the somatic nerves which came off from the same part of the cord as the sensory sympathetic fibres to the organ affected. Thus if I could map out the somatic areas along which pain was referred in visceral disease I could say, on Ross's hypothesis, what was the sensory supply from the sympathetic of the particular organ affected. By this means I obtained another scheme showing the distribution of the sensory sympathetic fibres analagous to that which Gaskell constructed for the motor and inhibitory fibres of the same system.

The next question that arose was whether these tender areas appeared only in visceral disease, and I soon found that they appeared to a greater or less extent in many general diseases, such as anæmia, ague and fever of all kinds.

An examination of the zones of analgesia or of tenderness in so-called hysteria showed that the sensory disturbances in this disease fall into two great groups—in one of which the areas of sensory disturbance followed the spinal type, whilst in the other the distribution was fundamentally different.

This opened up the whole question of sensation in its various forms, but in this paper I shall not do more than touch upon the relations between the distribution of the sensation of pain, of heat, of cold and of touch.

In the present paper I shall confine myself to the discussion of the disturbances of sensation, &c., below the level of the clavicles, for although every general statement applies equally to the head and neck and the body below the clavicles, the problems opened up by a consideration of the areas of tenderness occurring in the head and neck are so wide reaching that I must reserve them for a subsequent paper in the near future.

CHAPTER I.

GENERAL.

§ 1. Characteristics of the areas.

§ 2. The relation between the pain areas and the distribution of Herpes Zoster.

§ 3. Topographical.

§ 1. *The pain and areas of tenderness in visceral disease; their characteristics, and the means for their demonstration.*

In order to appreciate the characteristics of the areas of tenderness which appear in visceral disease it is best to take an example. In the type of case which is so common in our hospitals where a patient suffers from pain after food with or without hæmatemesis, an area of cutaneous tenderness is sometimes found which can be marked out as follows. The skin and subcutaneous tissues are taken up gently between the finger and thumb at various points on the abdomen. As soon as we pass a line extending from a point about two and a-half inches above the umbilicus to the tip of the eighth rib the patient at once complains of pain. On working from below upwards, tenderness is complained of as soon as a line is passed extending from the umbilicus to the tip of the tenth rib. The tenderness may lie entirely to the left of the middle line of the abdomen or there may be an exactly similar area on the right side.

If unilateral the tenderness is in the form of a triangle with the base at the linea alba, one side formed by a line with an upward slope from the umbilicus to the ribs, and the third side extending from the eighth rib to a point on the middle line of the abdomen two and a-half inches above the umbilicus. The apex is situated at about the ninth space. Now this anterior portion is associated with a posterior portion, also roughly a triangle. Its base is formed by the eleventh and twelfth dorsal spines, and its apex reaches a little further forward than the line of the posterior axillary fold.

A still better method of marking out such an area is to take a pin with a round head of such a size that it is obviously blunt to all parts of one's own cheeks and face. Travel over the surface of the abdomen using the blunt end only, in the same way as if you were using the point to test for analgesia. In a favourable case the patient does not complain until the limits of the tender areas are reached, when he at once complains that he is intensely sore and he will even cry out that you are pricking him. Thus in a favourable case the increased sensibility is so great that the contact of the blunt head of a pin is mistaken for a prick of the point. Subsequently in this paper this phenomenon will appear in my notes of cases as "head of pin called point." In a large number of cases there is no such confusion, but the patient says that, although he knows that the blunt end is being used, it gives him great pain. He feels "sore and bruised, as if he had been beaten about the body or back."

It is wonderful how unanimous patients are in their description of the sensation produced by touching such a tender area. I for one can fully confirm the description usually given by patients of their sensation; for such tender areas when touched, give exactly the sensation produced by touching a bad bruise. Many of my readers will have at one time or another suffered from these tender areas, and will be able to bear out my description.

I must warn my readers from supposing that the patient cannot tell the difference between the head and the point of a pin on these areas. Far otherwise. The touch of the head of a pin feels to him like a prick on a normal skin surface, whilst the point gives him acute pain far in excess of that caused by a prick upon the normal skin.

Another method for bringing out these tender areas, which is clumsy but theoretically interesting, is the use of a warm sponge or of a test tube filled with moderately warm water. As soon as the tender area is reached the patient experiences a feeling of great heat and flinches under the application. The same effect can be produced by moderate cold except that even moderate cold is at all times unpleasant to the skin of the abdomen, whilst moderate warmth is pleasant, and thus brings out the contrast between the normal and the altered sensory areas in a much clearer manner.

Another characteristic of these tender areas is the increased reflexes which can be obtained from them. This is a subject to which I shall return later, but I may mention here that whenever a portion of the skin becomes hyperalgesic from visceral disturbances the reflexes normally obtainable from that area are much increased. This is beautifully seen when the hyperalgesia affects the skin of the abdomen or buttock on one side only, so that the reflexes on the two sides can be compared with one another.

Immediately below the area of the abdomen which I have just described lies a tender zone which frequently appears in visceral disease. This area is bounded above by a line running from the umbilicus to the tip of the tenth rib and thence directly backwards to the first lumbar spine. Now supposing this sub-umbilical area is tender, no matter how intense the tenderness may be, its upper border never extends beyond this upper limit. Should the area which I have described above the umbilicus be marked out by tenderness its lower border never extends below the same line, however tender it may be. Again should the sensation of pain be entirely absent over either of these areas, in consequence of a destructive lesion to the spinal nerve roots or cord, we never find that the upper and lower limits of these two areas encroach upon one another *provided a prick is the test used for anæsthesia.* Thus it is evident that these areas do not overlap one another, and it is a peculiar characteristic of these areas of painful sensation that wherever they are situated on the surface of the body, whether they be marked out by hyperalgesia or analgesia, their limits never materially alter or overlap. Thus if we take any three areas adjacent to one another, of which the uppermost and the lowest are tender; the clear space between them of unaffected skin will represent the full extent of the middle area. This is both curious and unexpected; for we know that the areas on the skin supplied by the nerve roots[1] overlap considerably, and as a necessary result a circumscribed lesion does not produce a sharp border of anæsthesia to touch.

I have so far spoken as if a visceral lesion always caused tenderness over the whole of its appropriate area. This is not by any means the case. In disturbances of the stomach, instead of a whole tender area such as I have described above, we frequently find two tender spots, from an inch to an inch-and-a-half in diameter situated over the costal margin at about the tip of the ninth rib, together with a posterior spot generally situated near the eleventh and twelfth dorsal vertebræ, a little to one side of the middle line. Now, if the patient is asked where his pain is situated, he will point to one or both of these spots. Again, it is possible that he may suffer from pain only, without any tender spots on the skin, much less a whole band of tenderness reaching to the middle line in front. But even if cutaneous tenderness is absent, the pain is referred to the position of the maximum tenderness corresponding to the affected areas. Thus in cases of stone in the kidney, the maximum pain is situated at the tip of the twelfth rib, and at this point we can frequently demonstrate considerable cutaneous tenderness; this point obviously lies in the subumbilical area which I have described above. Every such area of cutaneous tenderness has one or more maximum points, the position of which is exceedingly important, for it is to the situation of these maxima that the patient refers his pain. Thus in each case, given the position to which the patient refers his pain, I can say to what area the pain belongs. For I recognise in these points to which the pain is referred the maxima of an area whose limits I have occasionally found marked out completely by tenderness. So fixed are these maxima, that supposing a whole area is tender, we can confidently predict that as the disturbance subsides, the tenderness will become limited to certain points, and when the disturbance has subsided still more, pain only will be left referred to the same points as the maxima. When I come to speak of the limits and relations of these areas, I shall therefore lay stress on the position of the maxima in each area as I describe it, for these maxima give us the clue to the meaning of pain referred to various spots on the body.

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[1] Sherrington, *Proc. Roy. Soc.*, vol. 52.

§ 2. *The relation between the tender areas in visceral disease and the areas affected in herpes zoster.*

Before I proceed to describe the limits of the tender areas in visceral disease, I must digress for a moment to point out how I have been helped in this investigation by certain trophic skin lesions, especially herpes zoster.

A controversy has long raged as to the true significance of herpes zoster, for it is at once evident to anyone who closely examines a case of herpes that the distribution of the vesicles does not in many cases follow the distribution of any peripheral nerve. As each case appears in the outpatient room, an attempt is made to show that it follows the course of an intercostal nerve, but such attempts mostly break down, and it is assumed that more than one nerve is affected. In Germany it has long been recognised that the eruption does not follow the line of any one nerve, and an attempt has therefore been recently made to explain its distribution by supposing it depends on the vascular system. This attempt, however, has not been satisfactory.

Now it has always been taught that herpes zoster is probably an inflammation of the posterior roots,[1] and that this explains the want of harmony between its distribution and that of the peripheral nerves. It therefore struck me that, if this were so, a careful collection of the areas occupied by herpetic eruptions would be an excellent method of mapping out the long sought distribution of the posterior roots. I therefore proceeded to collect cases of herpetic eruptions, and was astonished to find that they agreed in an extraordinary way with the areas of tenderness I had observed in visceral disease, for not only did the actual areas occupied by the eruption agree with the areas of tenderness, but I found that if the herpetic areas were carefully marked and measured, they were never found to overlap. Moreover the advent of the eruptions is mostly preceded by pain and tenderness of the skin. In three cases in which I was fortunate enough to see the patient before the eruption was developed, I was able to mark out an area on the skin which exactly corresponded to that seen in visceral disease; and I was unable at the time to say whether this area was due to reference from visceral disturbances, or was the percursor of a herpetic eruption. Again the herpetic eruption always makes its first appearance at certain points, from which in most cases it spreads. But even if the eruption spreads until it occupies the whole area, these points of first appearance are always the most affected. Now these points correspond to the positions of pain and maximum tenderness in visceral disease. Thus the areas occupied by herpetic eruption correspond to those which become tender in visceral disturbances in three points.

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[1] *Von Bärensprung Annalen d. Charité*, Bd. ix. 2, Bd. x. 1, Bd. xi. 2.

1st. They have the same distribution.
2nd. They do not overlap.
3rd. They have the same maxima.

Thus in the herpetic eruption I had a means of actually seeing the areas I was investigating, marked out for me on the surface of the skin.[1]

---

[1] I feel inclined to think that herpes zoster may be etiologically divided into two groups.

1st. The eruption may be secondary to irritation of the spinal cord, &c., by Caries or other nervous diseases, such as tabes dorsalis, or may be due to some nerve irritant such as arsenic.

2nd. The type of herpes which represents an acute specific. The onset of the illness is preceeded by 5 to 7 days fever thirst, loss of appetite, &c. The illness may begin with a rigor, or in a child with vomiting. The temperature falls with the outbreak of the eruption. The position of the eruption may, in some cases, be determined by long continued irritation, such as chronic cough, chronic intestinal catarrh, &c., but in many cases no such predisposing cause can be traced.

The herpetic eruptions which I shall use to illustrate this paper, are selected from a collection of sixty-two cases, all of which lay below the eighth cervical segment. Each case was drawn by myself from at least three points of view, and from a quarter to half-life size.

I cannot now enter into the question as to how far other skin diseases throw light on the distribution of these areas. I have interpolated the above short explanation in order that I may refer to the distribution of herpetic eruptions when we are discussing in the next section the limits of the areas of tenderness in visceral disease.

§ 3. *Topography.*

In the previous sections, I fear I may have led my reader to suppose that it would be an easy task to map out the areas of tenderness in visceral disease for the whole body. This is, however, not the case in practice. The first difficulty that meets us, is the only partial appearance of the areas in the forms of maximum tender spots. Now for these maximum spots to be of value, it is necessary that the whole area to which they belong should have frequently come under the notice of the observer, and yet it is only in a small proportion of the cases that he has the chance of seeing the area in its full development.

Then again, it is rare for one area only to be tender. In most cases, several areas are present at once; however, we frequently find that of three areas adjacent to one another, 1 and 2 will be present on one occasion, 2 and 3 on the next, and thus the limits of the three areas can be judged by exclusion. In fact, those cases where one area only is tender occurs so seldom, that if we had to depend upon such cases, many hundreds would be necessary before one could be found to satisfy our requirements.

But this leads me to speak of another difficulty which is by far the most formidable if we depend only on the areas which are produced by visceral disease. There are certain areas on the body which are extremely rarely affected. If they should be tender, they mostly appear in combination, so that we are unable to split them up into their constituent elements. Thus of five neighbouring areas, 1 and 2, 2 and 3, or 1, 2, and 3 may be present together; but if 4 and 5 appear, they are never present separately, but always in conjunction with one or more of the other areas.

It might be asked, how do we know that the tract of skin which we assume to be composed of several areas is not in reality a unity? The answer to this question will be given in full later when we speak of organic disease of the nervous system; for in nervous diseases, we find those tracts which we cannot differentiate by means of visceral disease split up into their constituent elements. Again, the distribution of herpes zoster helps us in a few cases to break up these compound tracts into their elements.

I shall now give the limits of each area with its characteristics in detail, and in every case I shall give:—

1st. The area of tenderness.

2nd. The maximum spots.

3rd. The position of the pain.

4th. The distribution of a case of herpes (when possible), which corresponds to the area described.

Though I give bony points as the guides to these areas, I must warn my readers that they are not absolute guides; for the only true guides to these skin areas are skin points, of which, unfortunately, there are very few. Thus the umbilicus is a much safer guide to skin areas than any bony point. Men, women, and children differ to such an extent from one another anatomically, that no actual measurement from, or dependence on bony points will give us the true nature of a skin area; and although its mutual relations render its nature obvious, we should go astray if we were guided by its relation to bony points.

Subscapulo Inframammary.

The first area which I shall consider runs the course seen in fig. 1. Its upper border starts behind at about the sixth dorsal spine, and runs forwards under the angle of the scapula to a point in front in about the fourth space. The lower border starts from about the eighth spine, and swings forward to the lower part of the sternum just above the base of the ensiform cartilage. This area cuts the eighth rib at the angle of the scapula; the seventh rib at about the level of the posterior axillary fold; the sixth rib at the anterior part of the axilla; the fifth rib in the nipple line.

The maximum points of this area consist of a spot under the posterior border of the scapula at about the level of the seventh dorsal spine, and about one-and-a-half inches from the middle line of the back; in front there is a well marked spot about the fifth rib, an inch internal to the nipple line.

The pain corresponding to this area, is said to be "between the blade bones" behind, and "just under the breast," or if situated on the left side, "just under the heart."

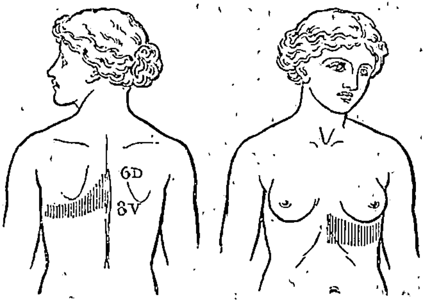

FIG. 1.

The shaded area represents the extent of the cutaneous tenderness in Case 1.

6D is placed at the level of the 6th dorsal vertebra. 8V at the level of the 8th dorsal spine.

Case 1.—Mrs. L. M., aged 35. Admitted Sept. 22. About two-and-a-half months pregnant. For rather over a month, constant vomiting. On admission to U. C. H., thin and wasted; constant vomiting and retching; pain under angle of scapula and under breast; cutaneous tenderness as in fig. 1. Sometimes right side, sometimes left side; sometimes more or less on both sides over this area; no tenderness elsewhere. Tongue furred; breath foul. No physical signs in heart or lungs. Pulse 96. Temp. 99.2. Urine normal. Erosion on cervix.

Twice much improved; relapsed again, and each time cutaneous tenderness reappeared. Ung. cocaine c. lanolin relieved pain and cutaneous tenderness, but did not stop vomiting.

October 23. Uterus cleared out.

November 9. Absolutely well. Has put on nearly a stone in weight.

A not uncommon form of herpes zoster is shown in the annexed fig. 2. It is never very marked over the spine, but a few spots are sometimes present over the sixth and seventh dorsal spines. There is always a well marked crop of vesicles over an area just internal to the angle of the scapula. In a well marked case the chain of vesicles run forwards, cutting the seventh, sixth, and fifth ribs, ending generally with a particularly well marked crop in the fifth space just internal to the nipple line. Sometimes two spots only are present corresponding very well with the maxima above mentioned. Thus we can see how closely this eruption corresponds with the area of tenderness. Moreover, in one of these cases, I was fortunate enough to be able to mark out an area of cutaneous tenderness of an intense character over this area before the herpetic eruption appeared.

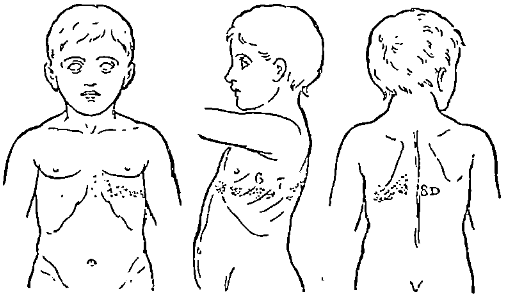

FIG. 2.

To show the distribution of the herpetic eruption in Case 2.

Case 2.—William S., aged 8. February 14.

Has complained for nearly two weeks that he could not raise his arm owing to pain in axilla.

Ten days ago, vomited. Went to bed with pain in the head and a sick feeling, but was well again next morning.

Four days ago eruption came out (fig. 2).

No surrounding tenderness.

Small glands in axilla.

Always has a slight cough and running at the nose. No physical signs.

Bowels open daily. No pain in stomach.

Here we have a typical case of zoster which runs round the body as a girdle crossing the line of several ribs, and not definitely following the course of one intercostal nerve.

The subscapulo inframammary area is characterised by

(1st) Two maxima, one just inside the angle of the scapula, the other over the fifth space under the nipple.

(2nd) Pain "between the shoulders" and "under the heart" on the front of the chest.

Subscapulo Ensiform.

This area consists roughly of two portions; a dorsal and a ventral triangle with their apices slightly separated from one another. It continually appears, especially on the left side, in stomach affections. [Fig. 27, p. 68.]

The base of the posterior portion is formed by the space between about the tip of the seventh or eighth spines and the nineth or tenth spines. Its upper border is formed by a line curving forwards about one and a half to two inches below the angle of the scapula to a point in about the seventh space at the level of the posterior axillary fold. The third side of the posterior portion is formed by an almost horizontal line from about the ninth spine forwards to meet the upper border just at the level of the posterior boundary of the axilla. The anterior half of this area is roughly a triangle, whose base is formed by a line extending from the base of the ensiform cartilage for from 2 to 2½ inches along the linea alba. The upper border is a curved line from the base of the ensiform along the lower border of the sixth rib to a point in the sixth space, just behind the level of the anterior axillary fold. The third side of this anterior triangle is formed by a line extending from a point in the linea alba 2 to 2½ inches below the base of the ensiform, directly outwards and backwards to meet the upper border in the axilla. Thus the apices of these two roughly triangular patches are separated from one another apparently by a distance which is a little greater than the breadth of the seventh rib.

The most common maximum point for this area is the well-known "epigastric spot," exactly over the ensiform cartilage. The posterior maximum is situated close to the middle of the back, at the level of about the eight and ninth dorsal spine, and extending outwards to the line of the angle of the scapula.

The patient locates the pain corresponding to this area "over the pit of the stomach," *i.e.*, over the ensiform cartilage, and over the centre of the back at about the level of the eighth dorsal spine.

These are the well-known places to which pain is referred in so many cases of gastric ulcer.

I shall not spend much time in describing the herpetic eruption which corresponds to this area, for a glance at the rough figure I give of a case (fig. 3), shows how closely it resembles the area on fig. 27.

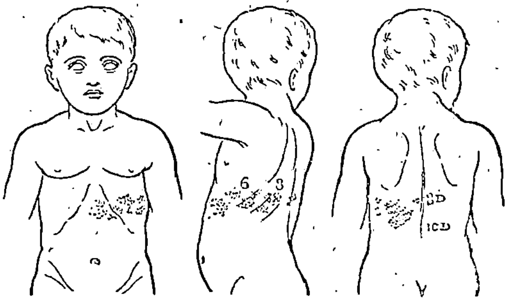

FIG. 3.

To show the distribution of the herpetic eruption in Case 3.

Case 3.—Harry P., aged 5, Nov. 9.

For two weeks has had a very bad cough. Feverish, hot and thirsty. No vomiting, except with the cough, which is so violent that he "heaves" with it.

November 4. During the night was very fidgety, could not sleep and complained of pain.

November 5. Spots began to come out. The patch in front came first. The posterior patch only came out yesterday.

Bowels regular.

Thus the subscapulo ensiform area is characterised by

(1st) Two maxima, one at the pit of the stomach, exactly over the ensiform cartilage, the other extending from the middle line of the back outwards to the angle of the scapula.

(2nd) Pain "at the pit of the stomach" and "just inside the shoulder blade."

Middle Epigastric.

This is a very puzzling area, and gave me a great deal of trouble before I could come to any conclusion with regard to its boundaries. It is particularly difficult to differentiate, owing to the fact that it is rarely marked out alone by tenderness in visceral disease. It most commonly appears with the one below it. It is, therefore, best made out by the method of exclusion, but I am fortunate enough to possess several cases where it appeared without its immediate neighbours. [*Vide* fig. 30, p. 74.]

The most important portion lies on the anterior surface of the body. Its upper limit consists of a line which starts from a point 2 to 2½ inches below the base of the xyphoid and passes upwards and backwards over the costal margin along the seventh rib. Thus far the upper border slopes upwards and backwards. But the remainder of the upper border of this area runs a different course. It starts from a point between the ninth and tenth spines, and runs horizontally out across the back, to a point about 2 to 3 inches from the middle line, just outside the line of the angle of the scapula. It almost touches the lower border of the subscapulo mammary (v. p. 74) area as it swings round the angle of the scapula. From this point the upper border rounds off downwards, just meeting the upper border of the anterior portions on the eighth rib.

The lower border behind is about one vertebra lower, if as much, and runs out parallel to the upper border until the angle of the scapula is reached, when it turns down to round off like the upper border.

The lower border of the anterior half of this area runs upwards and backwards from a point on the linea alba, about 2 to 2½ inches above the umbilicus, reaching the costal margin at the lower border of the eighth rib, and bending up to meet the upper border at about the middle of the axilla. Now fig. 30 shows very clearly the meaning of this curious conformation. For the blank spaces lying between the two shaded areas exactly represents the tract occupied by the subscapulo ensiform area (p. 13).

However it is rare to find the area marked out with anything like this precision, for its dorsal portion is so insignificant that it is usual to find the anterior or abdominal portion well marked, but the posterior part only represented by the important maximum in the line of the angle of the scapula.

The more important maximum belonging to this area is the one situated about 1½ to 2½ inches below the level of the angle of the scapula and from 2 to 3 inches from the middle line. The second well marked maximum is situated in the eighth space, about 2 inches outside the nipple line and almost in the mid-axillary line.

The pain of this area is the well known "pain under the blade bone" behind and over the eighth space towards the anterior part of the axilla in front. Both points of reference are well marked in liver disorders, especially on the right side.

I give a figure (fig. 4) of a case of herpes zoster following this area very beautifully. The measurements were somewhat small, for the patient was only eleven years of age. This figure illustrates the difficulty of relying on absolute measurements, for the figure (fig. 4) obviously corresponds to the area in fig. 30, but all the measurements were slightly smaller.

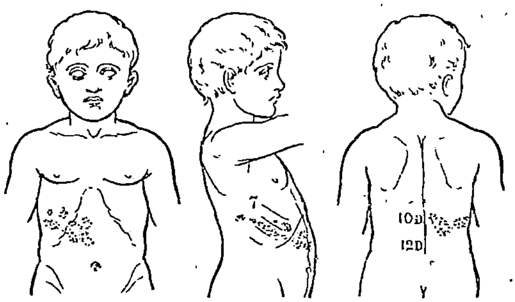

FIG. 4.

To show the distribution of the herpetic eruption in Case 4.

7 is placed over the 7th rib, and the sloping lines below it represent the 8th, 9th and 10th ribs.

10D represents the 10th dorsal vertebra.

Case 4.—Joseph S., aged 6, Oct. 26.

Has had a cold for a week, but eats well and is said not to have been "feverish."

Oct. 25th he showed the spots to his mother. She does not know which came out first.

No cough. Subject to bronchitis. Signs of emphysema with a few râles at the right base. Nothing definite in the lungs otherwise.

No vomiting. Bowels open daily.

Thus the middle epigastric area is characterised by

(1st) Two maxima, one in the 8th intercostal space, just external to the nipple line, and one about 2½ inches (in an adult) below the angle of the scapula.

(2nd) Pain "in the side," in about the 8th space, and pain "below the shoulder blade."

Supra-umbilical.

This area is simpler to describe than those which have preceded it. Its upper border extends from about the tenth or eleventh spine almost directly outwards across the base of the eleventh, tenth, ninth and eighth ribs. When nearly at the costal border it dips downwards to a point about 2½ inches above the umbilicus in the middle line. The lower border is formed by a line from the umbilicus upwards and backwards to the tip of the tenth rib; it then becomes a little more horizontal and runs almost straight back to a point corresponding about with the twelfth dorsal spine. [*Vide* fig. 26, p. 67.]

This area has a very well marked maximum point situated over the costal margin at the tip of the ninth rib (*i.e.* the tip of the tenth costal cartilage.) It lies partly on the costal border and partly just laps over to the abdomen and thus, when on the right side, exactly corresponds to the position of the gall bladder. The posterior maximum is not so prominent, but seems to lie at about the level of the eleventh dorsal spine about 1½ inches from the middle line of the back. Thus when double the patient complains of pain over the "middle of the back," and points to about the region of the eleventh spine over an area almost the size of the palm of the hand. In front the pain is always complained of as "just on the edge of the ribs." When on the left side this pain "over the edge of the ribs" is a very common symptom of gastric disorder.

Herpes is not at all uncommon over this area. Fig. 5 shows a fairly well marked case in which the posterior portion is somewhat more marked than usual.

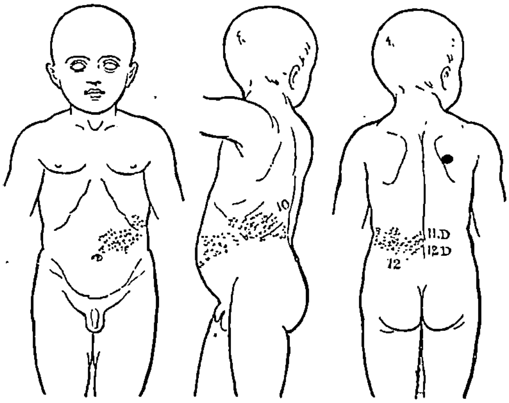

FIG. 5.

To show the distribution of the herpetic eruption in Case 5.
The eruption just reaches the umbilicus in front.
10 is placed over the 10th rib. The line above it represents the 9th, the line below it the 11th ribs.

Case 5.—Henry S., aged 3, Oct 28.

Very feverish, restless and irritable for a week.

Oct. 23, vomited, and again on Oct. 24. Since then his appetite has been very bad. Usually ravenous and can't be satisfied.

Bowels chronically out of order. Sometimes goes two or three days without a motion, and then many times in one day. Motions offensive. Rickety.

Cough. Bronchitis. A few râles over both bases. Rickety chest.

Oct. 28. Much better now the eruption has come out.

Thus the supra-umbilical area is characterised by

(1st) Two maxima, one over the tip of the 9th rib at the costal border and one over the back at the level of about the 11th dorsal spine, extending for a short distance outwards on the affected side.

(2nd) Pain "over the edge of the ribs," and over "the middle of the back," at about the level of the 11th and 12th dorsal spines.

Conclusions so far.

From what I have already said about these areas certain important conclusions can be drawn.

In the first place the areas of tenderness which appear as a consequence of visceral disturbance do not overlap. For if they overlapped to any considerable extent, like the areas of common sensation, the extent of skin covered when any one was present must necessarily be greater than that left unaffected when the areas on each side of it were tender. That is to say—supposing No. 1 and No. 3 were tender the skin between their borders, which was unaffected, must of necessity be of smaller extent than that affected when No. 2 only was tender. But this is not the case as is seen by comparing fig. 27 with fig. 30. The whole distance from the base of the xyphoid to the umbilicus is some 6 to 7 inches. Now of this distance the supra-umbilical area takes from 2½ to 3 inches, the mid-epigastric 1½ to 2 inches, and the subscapulo ensiform about 2½ inches along the linea alba. If the supra-umbilical area only is affected there is found some 4½ inches of unaffected skin between the base of the xyphoid and the upper border of the affected area; whilst if the two upper areas are affected the lower border of the affected tract of skin is found to lie some 2½ inches above the umbilicus. Thus if the areas overlap they overlap to such a small extent as to be within the errors of clinical observation.

We are on more difficult ground when we examine the records of herpetic eruptions, for so many cases of herpes zoster occur in children and young adults that the absolute measurements give no clue to the position of the eruption. We are thus obliged to fall back on careful drawings not made on a plan, but with the whole figure drawn in proportion from nature and marked with the bony points and relative measurements. We can then say that although many cases of herpetic eruption do not occupy the whole area we should expect, none of them transgress the lines I have already laid down, and absolutely none lie at an angle to or partly inside and partly outside the lines of the path areas. This last point is particularly interesting, for most of the areas of visceral tenderness lie *across* the line of the ribs; in this they resemble the distribution of herpes zoster.

In order that this paper may not be too long I shall now describe the lower areas more in tabular form speaking only in full of those which present some difficulty.

Sub-umbilical.

This is an easy area to map out for it occurs more often alone than perhaps any other area in the body. [*Vide* fig. 28, p. 71, and fig. 34, p. 85.]

The upper border is a slightly sloping line passing round the body from the first lumbar spine to the umbilicus. The posterior border is formed by the first to the fourth lumbar spines, whilst the anterior border extends from the umbilicus to a point about 2½ to 3 inches below it in the middle line of the abdomen.

The lower border is very curious for it is not a horizontal line like the upper border, but shows a curious downward tag—the first indication of the profound alteration produced by the limb. The posterior portion of the lower border extends from the fourth lumbar spine directly forwards and downwards across the extreme upper part of the crest of the ilium to a point just posterior to the anterior superior spine. Here the lower border takes a sharp turn upwards and backwards as high as the level of the crest of the ilium. From this point it again goes directly forwards and downwards to the linea alba 3 inches below the umbilicus. Thus taking the sub-umbilical area altogether, it forms a broad band extending round the body between the lower ribs and the crest of the ilium with a curious tongue, which is so arranged as to miss the whole of the anterior superior spine of the ilium.

This area has two well marked maxima, one of which is situated over the position usually occupied by the tip of the twelfth rib, the other over a point in front internal to the anterior superior spine of the ilium and about 1½ inches outside and 1 inch below the umbilicus.

The pain corresponding to this area is referred to the loins behind, over the tip of the twelfth rib and in front to a point in the iliac fossa somewhat external to and about on a level with the umbilicus. This is the area over which pain is referred in the cases of renal calculus. Patients suffering from this disease walk with the hand over the side affected, so placed that the thumb presses on the loins behind and the fingers on the iliac fossa in front.

Herpetic eruptions are common over this area and I have chosen a very beautiful one from my collection which is complete and shows the notch in a very perfect manner.

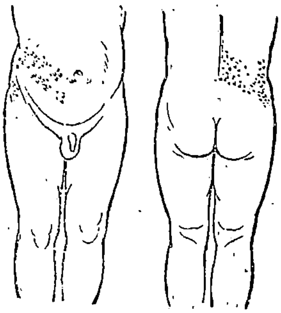

FIG. 6.

To show the area occupied by the herpetic eruption in Case No. 6.

Case 6.—William P., aged 4, Oct. 2.

No definite history could be obtained excepting that the eruption came out on Sept. 30. Bowels and appetite good. No cough.

Sacro-iliac.

This area is exceedingly difficult to obtain pure. Its maxima are constantly appearing in pelvic affections, but so soon as the whole area becomes tender some other neighbouring area is almost certain to appear more or less.

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The case I have selected for fig. 35 is interesting in that on the left side, the area that we are about to consider is alone tender, whilst on the right side it is associated with the sub-umbilical area which I have just described. As will be seen from fig. 35 this area has almost lost its resemblance to a band owing to the disturbance produced by the limb. It may be divided into a posterior and an anterior portion. Posteriorly it reaches as high as the spine of the fifth lumbar and the descending flap on to the buttock occupies roughly the sciatic notch and extends about 2 inches below the level of the highest point of the crest posteriorly. Anteriorly this area forms a simple iliac and inguinal flap reaching the middle line of the abdomen about 2½ inches above the pubes. It does not supply the part covered by the pubic hair in either male or female. There is also the tendency to the formation of a slight lateral flap. This cannot be seen on anterior or posterior diagrams, but requires a diagram of the side view of the body.

It is very interesting to notice that this area completes the one above, filling in the fork completely.

The two maxima belonging to this area are well marked and of frequent occurrence in pelvic disorders. Posteriorly we mostly find a spot of tenderness over the fifth lumbar and first sacral spines, extending outwards for at least 2 inches from the middle line of the back. In front the maximum is situated just above the position of the internal inguinal ring.

The pain corresponding to this area, is the "pain at the lower part of the back" (*i.e.*, over the fifth lumbar and first sacral spines), which so constantly occurs in women, especially during labour. It shoots round the hip "to the groin" (patient places his hand almost over the internal ring). If the pain is not sharp and shooting, it is fixed "in the lower part of the back and in the groin."

I have only once seen a nearly perfect herpetic eruption over this area (fig. 7), for most of them are more or less incomplete. Some are incomplete posteriorly; some anteriorly. I give a figure of the one which appeared to me to be most nearly complete of any I have yet seen.

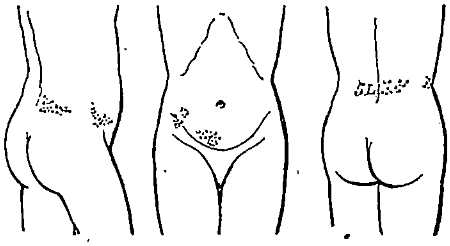

FIG. 7.

To show the area occupied by the herpetic eruption in Case No. 7.

Case 7.—Clara W., aged 12, December 21.

She has had pain over the lower part of her stomach for some time.

December 20. Eruption came out.

There is well marked cutaneous tenderness over a somewhat wider area than the extent of the eruption. This is so tender, that the head of a pin is called the point.

I also give the notes of an incomplete case [No. 8] for it was the case in which I first saw a complete area marked out by tenderness before more than a few vesicles had appeared. I was asked to see a case in the wards who was suffering from intense pain over the right side low down in the abdomen, and I discovered he had a very few herpetic vesicles. I could not then determine to what area they belonged, but mapped out the cutaneous tenderness, which followed very closely the limits of the area we are now discussing. The herpetic eruptions gradually developed and although it never became quite complete, it marked out the greater part of the area on fig. 7.

Case 8.—James H., aged 40. U. C. H. (Dr. Bastian).

Bronchitis and asthma for several years. Fresh attack, with swelling of the legs, five or six weeks ago.

On admission.—Marked emphysema; mucous râles all over the chest, with crackling râles at both bases. Tongue fairly clean. Spleen and liver not felt. No piles. Bowels usually regular.

Urine, normal.

Nothing cardiac.

November 25. Did not sleep owing to severe pain in right loin.

November 27. I found he had a band of intense cutaneous tenderness, marking out the sacro-iliac area on the right side. A few small herpetic vesicles have begun to appear on the abdomen.

November 28. Temperature rose for the first time to 100.6. Frontal headache. Pains in arms and legs. Shivering. Tongue furred. Appetite poor.

December 1. Herpes has developed fully.

Sacro-femoral.

This is one of the hardest areas in the body to deliminate, for it rarely, if ever, occurs alone, and its boundaries are so complicated, that even if it were present, it would take a long time to mark them out at all carefully.

It occurs most constantly with the sacro-inguinal area just described, and the case fig. 31 shows the two areas at the same time. Posteriorly, it causes an increase in the downward flap on to the buttock, so that this flap reaches quite 4 inches downwards from the level of the highest point of the crest of the ileum. In addition, there is a second great lateral flap extending to the top of the trochanter, and still further forwards it forms a triangle on the front of the thigh, reaching from 3 to 4 inches below Poupart's ligament. It seems curiously enough to supply a very small lateral flap on each side of the scrotum, the greater part of the pubic hair in the male, and a very small spot at the root of the penis.

There seem to be two maxima belonging to this area, one of which is situated just below the crest of the ilium, about 4 inches from the middle line of the back, and a second maximum which is always well marked over the outer border of Scarpa's triangle, just below Poupart's ligament.

The pain corresponding to this area is said to shoot from the upper part of the buttock, round the crest to the upper and inner parts of the thigh. Such is the account given by women after labour in whom this area appears as "after-pain." When the cause of the pain is continuous as in acute epididymitis, the pain is described as aching. [Compare fig. 32, p. 80.] This pain is situated about 3 to 4 inches below the level of the crest of the ilium behind, and in Scarpa's triangle, close to Poupart's ligament in front. One of my patients said it was as if someone had knocked him "in the groin, and in the back," the pain was the dull aching pain of a bruise.

I have seen but two cases in which this area only was marked out by herpes zoster. As far as I have yet seen, it has nearly always appeared in conjunction with the area above it, excepting only when it was very incomplete. I give [fig. 8] one of the two cases in which this area appeared alone, and at the same time was fairly complete.

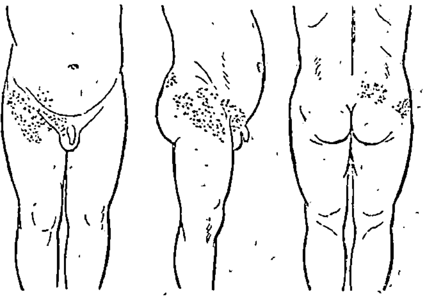

FIG. 8.

To show the area occupied by the herpetic eruption in Case No. 9.

Case 9.—William C., aged 5 (Dr. Crocker), December 23.

December 15. There was not a spot upon his body, and the child seemed perfectly well.

December 16. He was hot and feverish; wanting to drink water, but did not lie about or seem very ill.

December 18. The eruption began to appear as small blisters. No cough. No vomiting. Bowels daily. Quite normal. Appetite poor. Passes water well. No physical signs.

Gluteo-crural.

This area appears frequently in visceral disturbances but practically never alone. Its lower limits are easily defined for it is the last of this set of areas on the front of the thigh to appear in visceral disease. The case I have figured [fig. 36, p. 89], was one in which there was some catarrh of the cervix uteri. The cervix was dressed with copper and a Playfair's probe passed. As the result of the treatment she showed the tenderness I have figured, which passed off in a few hours. There was in addition a small spot corresponding to one of the sacral areas. The area figured was present on both sides. This I take to be a case in which by a lucky chance this area above of all the lumbar areas was affected and it shows what I take to be its limits fairly well. It does not seem to reach the middle line behind but its most posterior point lies three inches from the middle line just at the level of the fork of the buttocks. This area shows to a greater extent than any we have yet seen the alterations produced by the limb; for here both the lateral and the anterior flaps are pulled out on to the limb to an enormous extent. It has two maxima; one especially well marked just above and to the inner side of the knee and one situated close to the great trochanter. The pain will be described more fully later on, and at present it will be sufficient to say that the great feature of the pain corresponding to this area is its shooting across the thigh to the inner side to end above the knee.

I have only once been fortunate enough to see this area marked out completely by herpes zoster (fig. 9). It mostly occurs either with one or two areas above it.

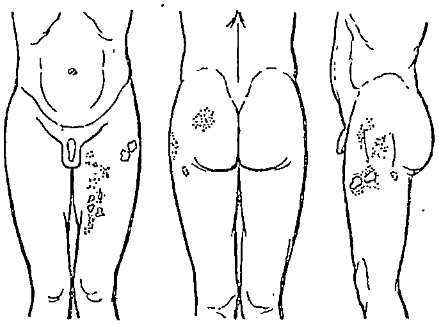

FIG. 9.

To show the distribution of the herpetic eruption in Case No. 10.

Case 10.—William E., aged 11, Oct. 16.

Eruption came out Oct. 2.

A week before this he suffered from headache and pain in the stomach. He shivered and complained that he was cold and had pains all over.

Then pain "at the side of his privates" came on and finally pain over the front of his thigh.

Weak, weedy looking child. Appetite bad. Bowels open every day. No cough. No physical signs in chest.

From this point downwards to the sole of the foot there are no areas which become tender in consequence of visceral disturbances excepting, as I shall show later, when some great change has taken place leading to universal tenderness of the whole leg. Thus from the visceral aspect the remaining skin in front of the thigh and leg is untracked desert. I shall therefore pass on to the posterior aspect of the thigh.

Sacral Areas.

The area of the skin now to be considered comprises the Perineum, Scrotum and Penis, a portion of the buttock and posterior aspect of the thigh as far out as the posterior border of the external hamstrings, the upper median portion of the calf and the sole of the foot. The areas which make up this tract of skin appear with decreasing frequency from above downwards, and if one of the lower ones is present, it is practically always accompanied by one of the areas above it. Thus I cannot give a case of visceral disease to shew each area separately but must show how the addition of each area alters the extent of the total skin affected.

Fig. 33 shows a tract of skin which is not uncommonly tender in painful affections of the mucous membrane of the bladder. The case from which the figure was obtained was that of a man from whose bladder a piece of mucous membrane was accidentally removed in the jaws of a lithotrite. In a second case an exactly similar area of tenderness appeared after the bladder had accidentally been washed out with 1 in 20 carbolic acid instead of with a harmless lotion. This tract of skin when bilateral is characterised by a small patch of tenderness over about the third or fourth sacral vertebra closely adjoining a saddle-shaped area on the back of the thigh; it extends into the perineum and covers the whole of the scrotum and penis excepting at the extreme root where it meets the sacro-inguinal area.

It will be noticed that I have coloured small portions of this tract differently in plates I. and II. I believe that the extent of skin shown in fig. 33 is in reality made up of two distinct visceral areas but as they almost always occur together it is exceedingly difficult to separate them. However, I believe that in some cases of prostatic disease the area marked S 4 on plate IIA, extending forwards to a point just in front of the anus, was not affected. In a case of stone impacted in the ureter it certainly was not tender whilst the front part of the perineum was sore to the touch.

Thus I believe we have here to do with two areas one of which is so small that it may easily be missed and is situated over the sacrum reaching as high as a point corresponding to the second sacral spine. It passes forwards over the coccyx to the skin around the anus. It also in all probability takes in the inferior half (or even the whole) of the glans penis with a small portion of the inferior surface of the skin of the penis.

The remainder of the tract of skin figured (fig. 33) corresponds to a second area—the gluteo-pudendal, which occupies almost exactly the parts in contact with a chair in sitting. In addition it occupies the whole of the scrotum and the remainder of the penis.

In these two areas taken together there are three prominent maximum tender points, which constantly appear in disease of the pelvic viscera. The posterior one is situated over the lower part of the sacrum and possibly belongs to the sacro-anal area. Another very common tender point is situated almost exactly over the ischial tuberosity whilst a third is situated at the tip of the glans penis. Pain corresponding to these areas is said to come from the back and "shoot into the privates." The patient also complains of a feeling of weight and pain in the perineum and has an aching bruised feeling over the ischial tuberosities. He also complains of the pain over the glans penis so well known as a symptom of stone in the bladder.

I shall reserve a consideration of the areas mapped out by herpes until I have described all the areas of this group; for a herpetic eruption so seldom appears over one area only in this group that a preliminary knowledge of the distribution of this group is necessary for the due comprehension of the herpetic eruptions occurring in this region.

In profounder disturbances of the pelvic viscera we sometimes find an area marked out in the back of the thigh corresponding to the areas marked S 4, S 3, and S 2, on plate IIA. Now the whole of the upper portion of this tract obviously represents the sacro-anal and gluteo-pudendal areas. Subtracting these, a big strip remains down the back of the thigh about three inches in width, reaching to the upper part of the calf and extending outwards to the posterior border of the external hamstrings [marked S 2.] This area never appears alone in visceral disease.

It has a very well marked maximum point over the upper part of the calf of the leg, which is a prominent feature in many pelvic disorders; there is also a second maximum situated about the middle of the posterior aspect of the thigh.

The pain corresponding to this area is the pain in the calf, which is so troublesome in some cases of prostatitis. It is much increased by walking.

The next addition to this tract is easily defined, not because it appears alone, but because it is separated from the rest of its fellows. It covers the whole sole of the foot and invades the dorsum, as in plate IIA, S 1. A posterior flap runs up to a point on the calf about 5 inches above the level of the malleolar line.

The commonest tender point belonging to this area is situated on the sole of the foot about 1½ inches in front of the heel. There also seems to be a second tender point in the anterior part of the sole just on the outer side of the base of the great toe.

Both these points are the seat of pain belonging to this area. Patients also frequently complain of "aching in the soles of the feet," which prevents their standing for any length of time.

The last area of this group to be added to the tract of skin I have described occurs but seldom in visceral disease. It is situated on the outer side of the leg, and swings over to the dorsum of the foot. The whole coloured area in fig. 34, represents the extent of the tenderness in a case of prostatitis, probably caused by the passage of instruments for the first time. Now the area over the buttock is the gluteo-pudendal, the area on the back of the thigh and calf is the sciatic, the area over the sole of the foot the soleal, and we have a fresh addition on the outer side of the leg, and the dorsum of the foot. This area is never in any case affected alone, and rarely without the sole of the foot being first affected.

The characteristic tender points of this area are situated over the dorsum of the foot and outer aspect of the leg. It is to these points that patients refer their pain when this area is affected.

From this point onwards we again come to the gap which, as I stated above, is never affected in visceral disease. Thus this gap is now seen to lie between the gluteo-crural area on the lumbar side and the fibulo-dorsal area of the sacral group.

Herpes Zoster over the Sacral Areas.

Unfortunately herpes zoster on the posterior aspect of the legs is rare, and when it appears over one area it is particularly liable to spread to one or more neighbouring areas. Von Bärensprung[1] figures one view of a case where the eruption almost exactly coincided with the two upper sacral areas, as we saw them in fig. 33. This case I reproduce in fig. 10.

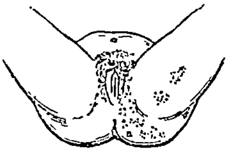

FIG. 10.

To show the distribution of the herpetic eruption in Case No. 11. [Copied from von Bärensprung, "Charité Annalen," Bd. ix., Heft 2, p. 101.]

Case 11.—From von Bärensprung, p. 101. Anna Rahn, aged 19. Taken in June 9th, 1861, on account of soft sores and bubo. Menstruation regular until eight weeks ago. June 11th began to menstruate. Menstruation ceased June 13th. In the night of June 12th she felt a burning sensation in the neighbourhood of the left tuber ischii, and the next morning a group of clear vesicles appeared on this spot. The eruption gradually increased until it occupied the area in the figure. She also had a patch on the buttock extending down the upper and inner part of the thigh for a short distance. Another patch lay just over the fold of the buttocks on the lower part of the sacrum. The praeputium clitoridis was affected.

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[1] *Loc. cit.*, Bd. ix. 2, p. 101.

This eruption seems to correspond exactly with the upper two sacral areas. The next one which I figure belongs to the next two sacral areas—the sciatic and the soleal. Neither area is quite completely represented, but I think the eruption is wide spread enough for us to say that it represents very fairly the soleal and sciatic areas. It is interesting to note how the gluteo-pudendal area is spared.

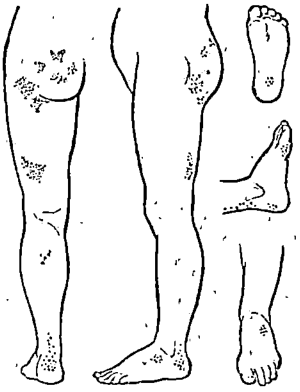

FIG. 11.

To show the distribution of the herpetic eruption in Case No. 12.

Case 12.—Harriet H., aged 5½, October 11.

Two weeks ago complained of pain and aching in the feet. This aching lasted 2 to 3 days and then the eruption came out as a spot in the middle of the thigh. A day or two afterwards the gluteal spots appeared. The eruption on the foot was not seen until a week afterwards.

Appetite bad for months. Sick in the morning for a long while. Nothing in the perineum or anus. Bowels open daily, but sometime ago they were very irregular. No cough. Nothing in the chest.

The pain was very bad until 6 days ago, and the leg was very tender. No tenderness now.

The Upper Thoracic Region.

We have now followed these areas of tenderness from about the level of the fourth rib down to the thigh, and from the sacrum down the back of the leg. I had intended to describe the areas for the head and neck, and thus work downwards through a second gap, which we shall find in the arm, to the subscapulo inframammary area with which we started. A consideration of the problems opened up by a description of the areas on the head and neck would, however, make this communication of such inordinate length that I shall treat of the head and neck in a subsequent paper.

The lower limit of the lowest cervical area which is affected in visceral diseases is shown in plate I. and II. A and B. Now between this line and an area on the ulnar border of the fore-arm I have never yet seen tenderness as the result of visceral disease. Thus we here find a second gap, which can only be filled in later when we consider the disturbances of sensation produced by organic disease of the spinal cord and roots.

Dorso-ulnar.

Cardiac cases frequently complain of pain down the ulnar side of the forearm. This pain is occasionally accompanied by cutaneous tenderness. Fig. 21 shows this tenderness in a man with aortic disease, who frequently suffered from paroxysms of pain. After one of these paroxysms had passed off he was found to have intense cutaneous tenderness, as in fig. 21.

This area has at least three maxima, the posterior of which is situated over the first dorsal vertebra and a little to one side of the middle line, the anterior over the second space and third costo-sternal junction close to the middle line of the chest. A third maximum is situated on the inner side of the forearm, just below the bend of the elbow.

The pain over this area is of an agonising character, especially in cardiac disease. It is said to dart through from the back to the front and at the same time to appear in the forearm, usually coming up from the little finger to the elbow.

I am able to give a most beautiful case of herpes exactly marking out this area.

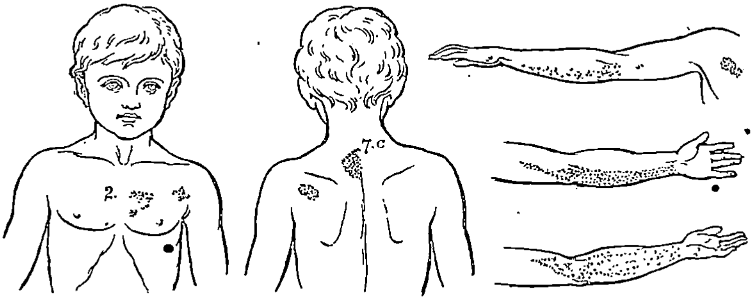

FIG. 12.

To show the distribution of the herpetic eruption in Case No. 13.
The figure 2 is placed over the lower border of the 2nd rib.
The figure 7c is at the level of the spine of the 7th cervical vertebra.

Case 13.—Henry George R., aged 7, August 17.

Eruption came out August 16. No antecedent pain. No cough. No physical signs. Appetite good. Bowels regular.

Dorso-brachial and Scapulo-brachial.

I can produce no case from my records in which each of these areas was separately marked out in its entirety. It is not at all uncommon in diseases of the chest to find the maxima of each area perfectly pure, but as soon as the whole of either area becomes tender it seems to bring the other with it.

It will be better therefore to consider the herpetic eruptions belonging to this group before proceeding to the tenderness produced by visceral disease. Fig. 13 represents a very complete specimen of herpes over the dorso-brachial area. The eruption started at about the level of the second dorsal spine and a very marked patch of considerable size lay between the spine of the scapula and the vertebral spines. The posterior and inner border of the arm was affected as far as the elbow. In front there were two patches in the third space extending just internal to the nipple line.

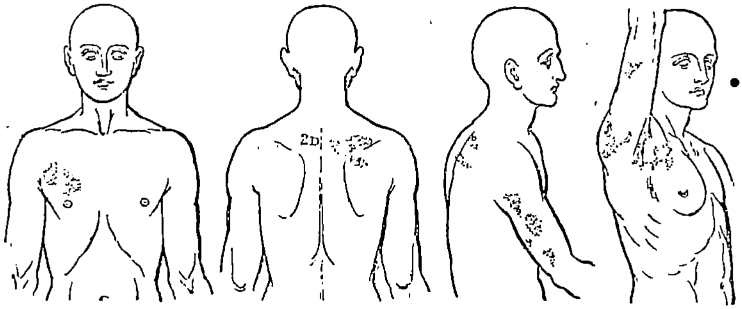

FIG. 13.

To show the distribution of the herpetic eruption in Case No. 14. Case No. 15 showed an almost exactly similar distribution.
2D is placed at the level of the spine of the 2nd dorsal vertebra.

Case 14.—James C., aged 27, carman, Feb. 11.

Feb. 10. Eruption first noticed. No antecedent pain excepting a slight feeling of soreness. In fact if he had not seen the eruption he would not have known that anything was the matter. Bowels quite regular. Appetite excellent. No cough. No physical signs. Never been ill in his life.

Very strong, healthy looking man with enormously developed muscles. Has been at work lately carrying very heavy wool-packs.

Case 15.—John R., aged 34, carman, Dec. 31.

For four or five days he had a feeling of weight over the third dorsal vertebra which made him constantly want to hold his head up. He frequently has this feeling in the back "whenever he has caught a little fresh cold." Having recently "caught a fresh cold" the sensation was not unexpected, but he was surprised to see the eruption come out on Dec. 30.

Eruption follows the same lines as Case 14, except that the arm is less affected.

Always has a cough, worst at night. No loss of flesh. Sibilant rhonchus at right apex in front, but at right apex behind distinct fine crepitations are audible.

Jan. 11. The herpes has gone. Considerable pain and tenderness over the second and third dorsal vertebræ. Also distinct tenderness over anterior part of axilla. Pain and cold feeling down the inner side of the arm. Fingers get bent up and numb, so that he cannot move them.

Note that he is a carman and has been engaged lately in carrying sacks.

Fig. 14 shows an eruption over what I take to be the third or scapulo-brachial area of the group. It reaches the middle line of the back about the level of the third dorsal spine. Its characteristic feature is the area over the subspinous fossa of the scapula, which resembles a small scapula in shape. In front the eruption extends on to the extreme inner border of the arm for a short distance and a few spots appeared in the third space internal to the nipple line.

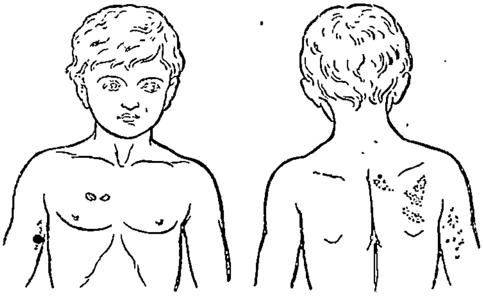

FIG. 14.

To show the distribution of the herpetic eruption in Case No. 16.

Case 16.—George C., aged 10½, Dec. 11.
Eruption came out Dec. 8. For three days previously he had pain over the area of the eruption.
Appetite poor for a week. Not feverish.
No cutaneous tenderness.
No cough. No physical signs.

Fig. 22 was obtained from a man who suffered from pain accompanied by cutaneous tenderness due to an aortic aneurism. The aneurism came forwards to the region of the second rib to such an extent that it was simply covered by skin. The area which it occupied is shown in the diagram (fig. 22A) and it was fortunately so situated that the referred tenderness did not correspond to the pulsating swelling. Now if we compare this figure with those showing the distribution of herpes (figs. 13 and 14) it is obvious that both areas are represented in consequence of the disturbance produced by the aneurism. But in addition a third area is present which I shall next describe.

The maximum tender points of the dorso-brachial or upper of these two areas are situated the one posteriorly exactly on a level with and over the more posterior point of the spine of the scapula and anteriorly in the third space exactly in and a little to the axillary side of the nipple line.

The characteristic tender point is situated posteriorly exactly over the centre of the scapula in the infraspinous fossa.

The pain of both these areas is said to pass through from back to front and to radiate down the arm; the one to the ulnar side of the elbow, the other to about the middle of the arm.

The Dorso-axillary.

This area is well shown, in plates I. and II., D 4. It is of frequent occurrence in lung disease.

The maximum tender points of this area are situated the one just internal to the posterior border of the scapula, the second over the posterior axillary fold and the third at the nipple, which always feels painful when this area is affected.

On fig. 15 we see another form of herpes occupying what I take to be the fourth area of this group. It starts from the middle line of the back at about the fourth dorsal spine, and swings outwards and downwards across the scapula. It also affects the posterior axillary fold, and goes into the axilla. In front, it forms a marked area above the nipple, sending a projection down towards the nipple itself, which is not, as a rule, affected in this type.

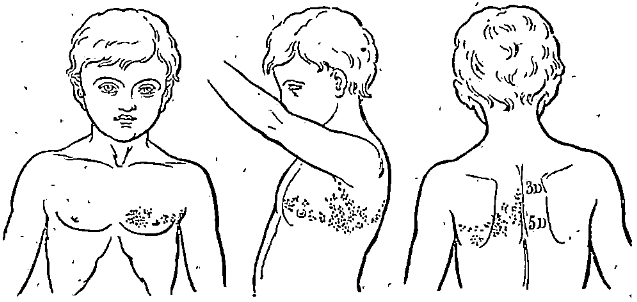

FIG. 15.

To show the distribution of the herpetic eruption in Case No. 17.
3D is placed at the level of the spine of the 3rd dorsal vertebra.
5D is placed at the level of the spine of the 5th dorsal vertebra.

Case 17.—Herbert D., aged 12, October 10.

He has been hot, feverish, and restless for three or four nights. Has a cough off and on for years. For the last 3 to 4 days the cough has been much more troublesome.

October 9. Eruption came out.

No pain. No tenderness.

Cough. No alteration in percussion note. Râles all down left side, and a few on the right base. Appetite poor. Bowels regular.

October 12. The eruption has developed, and now covers the whole area.

The Scapulo-axillary.

This area is frequently affected in all painful affections of the glandular tissue or ducts of the breast. Cracked nipples and over lactation will produce it, but it also occurs in men as I shall show later in consequence of malignant disease of the œsophagus. It is characterised by a dorsal patch close to the posterior border of the scapula at the level of about the fifth spine. It swings round the scapula to the axilla where it forms a marked flap, and ends in front in a patch just under the nipple. [*Vide* fig. 35.]

The maximum tender points are situated over the posterior patch, over the axillary patch, and over a point just on a level with, and internal to the nipple.

The pain is said to be "between the shoulder blades" and "just under the nipple."

A case of herpes belonging to this group is represented in fig. 16. It starts at about the fifth dorsal vertebra, and swings round the angle of the scapula all the vescicles lying above the border of the bone. In front, it is represented by a group of vescicles external to, and a group internal to the nipple, but the nipple itself remains quite free.

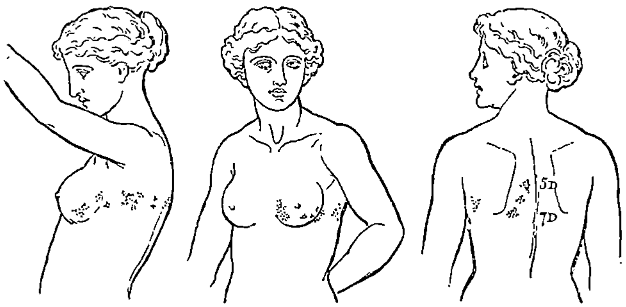

FIG. 16.

To show the distribution of the herpetic eruption in Case No. 18.

5D is placed at the level of the spine of the 5th dorsal vertebra; 7D at the level of the spine of the 7th dorsal vertebra.

Case 18.—Mary Ann L., aged 38, married, October 16.

October 1. Pain all up the left side. Shivering, cold, feverish. Loss of appetite. Bowels confined. Pain and giddiness in the head.

October 6. The eruption came out underneath the axilla. Pain continued till October 15, and she had great pain between the shoulders.

Has had eight children. Is now weak and run down from suckling a child of 17 months to prevent conception.

Now the next area below this is the one we started with, and is fully described on page 10.

Conclusions.

In this chapter I have described a series of areas which are tender in disturbances of the various organs. From the highest dorsal area (dorso-ulnar) I have traced 13 areas which fit into one another, and do not overlap to any appreciable extent. I have given cases of herpes zoster to show that the eruption in this disease appears over exactly the same areas as those which are tender in visceral disease.

We then passed to the buttock and the back of the leg, and marked out in a similar manner five areas belonging to the lumbo-sacral group. Between the lowest of the dorso-lumbar group, and the highest of the lumbo-sacral set is a gap which is not affected in visceral disease. This gap I shall fill in in the next chapter with the help of organic lesions of the nervous system, and a few cases of herpes zoster.

CHAPTER II.

THE SIGNIFICANCE OF THESE AREAS.

§ 1. To what level of the nervous system do they correspond?

§ 2. The numerical determination of the areas at their origin in the spinal cord.

§ 1. *To what level of the Nervous System do they Correspond?*

I have described the areas of tenderness occurring in visceral disease from the arm downwards and shall now attempt to determine the level of the nervous system to which they belong.

Now as far as visceral disease only is concerned these areas might correspond to a cortical or spinal distribution or they might represent the supply of posterior roots or peripheral nerves.

It is exceedingly unlikely that they bear any relation to cortical distribution for as I pointed out above they are identical with the areas of herpetic eruptions and they closely correspond as I shall show below to the areas of loss of sensation in organic lesions of the spinal cord.

Thus these areas which are tender in visceral disease must belong primarily to the lower level of the nervous system and must correspond to the distribution of spinal segments, spinal nerve roots or peripheral nerves.

Any one who has attempted to describe the distribution of a herpetic eruption in terms of peripheral nerves knows how difficult it is and how seldom the results are satisfactory. Take for instance the area shown on fig. 2. It starts behind at the back of the seventh spine, crosses the ninth space at the angle of the scapula and in its course to the front cuts the eighth, seventh and sixth ribs and finally ends partly in the fifth space.

Again the twelfth dorsal nerve passes over the curve of the ileum and supplies a skin flap closely resembling the trochanteric flap of the sacro-femoral area [p. 24]. But this in no way resembles the analgesia in a case in which the twelfth dorsal nerve was cut in an operation on the kidney. The borders of the resulting loss of sensation were not sharply defined, but one gradually passed from complete to partial analgesia. Moreover the area of analgesia partly occupied the position of at least two pain areas and covered neither completely.

The improbability that these areas correspond to the distribution of peripheral nerves is best shown on the limbs. For though in the upper limb the patch on the inner side of the arm evidently represents the intercosto-humeral the patch on the ulnar side of the arm back and front (dorso-ulnar area) running down to the little finger represents no peripheral nerve.

Again the gluteo-crural area corresponds fairly well with the supply of the external cutaneous and the internal cutaneous nerves from the lumbar plexus. But the middle cutaneous, which apparently is more ultimately associated with the internal cutaneous at its origin, is never affected.

Thus it is evident that these areas do not represent the supply of peripheral nerves.

Now if these areas do not represent a cortical distribution or the supply of peripheral nerves we have but two alternatives left. They must either correspond to the supply of spinal segments or to that of spinal nerve roots.

Ross held the view that the pain of visceral disease was referred along the line of nerve root distribution. But at the time his paper was written our knowledge of the supply of the nerve roots was more limited. Moreover he depended on the patient's account of his pain—a most unsatisfactory guide.

When I therefore discovered that the pain was associated with cutaneous tenderness it was at once evident that if Ross's ingenious hypothesis was correct I possessed in this tenderness a means of determining the supply of the various nerve roots by a physiological rather than by an anatomical method.

Now if we map out the distribution of the anterior primary division of any nerve root in the mid-dorsal region, and then add the distribution of the posterior primary division of the same root, the resulting tract will closely represent one of the areas I have described.

Moreover, von Bärensprung held that herpes zoster was an inflammation of the ganglion of the posterior root, and as we have repeatedly seen, there is a remarkable similarity between the areas occupied by a herpetic eruption and those which become tender in consequence of visceral disease.

Thus at first sight it appeared as if Ross's hypothesis were correct and that the areas I have marked out represented the distribution of the posterior roots.

But more than a year ago my friend Dr. Sherrington[1] communicated to me the results of his experiments on the distribution of the posterior roots, and pointed out that the areas of cutaneous supply from any two roots overlapped enormously. Thus on dividing one posterior root, Sherrington obtained no loss of sensation to touch, and he was obliged to adopt the following ingenious device, in order to discover the skin supply of any one root. If he wished to investigate the supply of the seventh dorsal root, he divided all the posterior roots from the first to the sixth dorsal, and from the eighth dorsal to the first lumbar. Thus he had left one root intact, dividing five roots above and five below. He was then able to mark out an area of sensation, bounded above and below by tracts of skin over which sensation was absent. The areas which he obtained by this method in the monkey, bear a close relation, as far as their position is concerned, to those which I obtained according to my method; but whereas his areas overlapped greatly, the areas I had marked out did not overlap at all.

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[1] *Proc. Roy. Soc.*, vol. 52.

Thus we at once meet with a difficulty in assuming that the areas I have marked out represent the supply of posterior nerve roots. The solution might be found in the following hypothesis:—

Sherrington's areas represent the root supply for the sensation of touch, whilst my areas represent a similar supply for the fibres subserving pain, heat, cold and trophic disturbances in the skin. And thus we might assume that whereas the distribution of the fibres for the sensation of touch in the various posterior roots overlap considerably, those for pain, &c., do not overlap.

But Sherrington expressly states that he used touch and pain stimuli indifferently as a test for the presence or absence of sensation, and thus the areas he marked out represented in all probability the supply both of the touch and pain fibres of one posterior root. Moreover, I have on five occasions, been able to observe the results produced by the surgical division of a single posterior root in man. There is of course a certain element of doubt about such cases, in that the division of the root occurred during an operation for disease. However, they all show complete absence of anæsthesia to touch after division of one posterior root. In most of the cases the sensation of pain, heat and cold was also not materially disturbed, but in some there was very slight and uncertain dulling of painful sensation.

Again, I soon found that the borders of the analgesia were much less definite in those cases where there was reason to believe that the roots were mainly affected, than in those where the spinal cord itself was mainly implicated. Thus my clinical experience, so far, leads me to think that the areas I have marked out do not represent the supply of an actual posterior root and I think we must assume that whereas Sherrington's areas represent the true root supply, my areas correspond to the supply, not of roots, but of segments of the spinal cord from which the roots in part arise.

If we confine our attention solely to the phenomena produced by visceral disturbance, we are obviously only permitted to assume that my pain areas represent the supply of portions of the spinal cord. For we must suppose that a sensory disturbance passes up the nerve fibres from the viscera to set up changes in the activity of that segment of the spinal cord in which they terminate; and that these disturbances of activity are referred to that portion of the skin which is supplied by the affected segment. And when we turn our attention to the results produced by localised organic lesions of the spinal cord, we find that the affected areas closely correspond to those which appear in functional disturbances of the viscera.

But this is only true for the sensation of pain, and does not hold for the sensation of touch, even when we have reason to believe that the substance of the cord itself is affected. On testing the majority of such cases it soon became obvious that the anæsthesia to touch had a very indefinite border. One gradually passed from the area where a touch was not felt, to one where the patient was in doubt. Thus the upper limit of the anæsthesia was not a definite line. On the other hand the upper limit of the loss of painful sensation was frequently quite definite. Thus I can but suppose that a large number of records of "anæsthesia," in cases of spinal injury, are in reality records of the loss of painful sensation or analgesia. In some of the cases in Thorburn's book[1] it is definitely stated that the loss to touch was not so marked as loss to pain; and Allen Starr[1] in his most interesting paper, definitely calls attention, in several of the cases which were under his own care, to this want of agreement between the loss to touch and the loss to pain. In every case the loss to pain was more extensive and more definite than the loss to touch. Thus I think we may assume that in most reported cases of spinal injury, unless touch and pain are specially noted (as in Starr's cases) the description of the "anæsthesia" corresponds in reality to loss to painful stimuli.

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[1] A Contribution to the "Surgery of the Spinal Cord," by Wm. Thorburn. London, 1889.

Now an examination of a series of cases of injury to the spinal cord itself brings out the close relation between the areas of analgesia produced by such injuries and the areas which are tender in visceral disease. It also shows that the central connections of the fibres for pain and those for touch differ considerably, for the loss to pain is nearly always more definite and more extensive than the loss to touch.

To sum up the results arrived at in this section, we must conclude that there is a fundamental difference between the areas marked out on the surface of the body by Sherrington and by myself in that, whilst his areas overlap considerably, mine do not overlap to any appreciable extent. Now whilst his areas undoubtedly represent the cutaneous distribution of posterior nerve roots, my areas, at least as far as they concern the tenderness produced by visceral disease, equally undoubtedly represent the cutaneous supply of segments of the spinal cord from which these roots in part arise. But examination of the records of traumatic lesions of the spinal cord shows that the disturbance of touch is less profound than the disturbance of the sensation of pain, heat and cold. Therefore the mechanism for the sensation of touch in the various spinal segments must overlap, whilst that for the sensation of pain, heat and cold does not overlap at any rate to the same extent.

Thus, in conclusion, we may, I think, assume that the areas which are tender in consequence of visceral disturbance and the areas occupied by the eruption in herpes zoster

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[1] *Amer. Jour. of Medical Science*, July, 1892, and also "Familiar Forms of Nervous Disease." New York, 1891.

represent the supply of the fibres for the sensation of pain, heat and cold, and of the trophic fibres to the skin from various spinal segments. They also roughly represent the regional supply of posterior roots; but probably the difference between the cord supply and the root supply will be found to lie in the absence of overlapping in the cord and the existence of overlapping in the root supply at various levels.

§ 2. *The Numerical Determination of the Areas at their Origin in the Spinal Cord.*

If the areas which I have described correspond to the cutaneous supply of the nerves for pain in each spinal segment we shall be able to assign to each area its proper number. But both in the arm and in the leg we are checked by a gap if we depend only on the areas of tenderness in visceral disease. Thus it is impossible to begin at the fourth cervical root and count downwards or at the last sacral area and count upwards.

Fortunately however, we are able to learn the origin of a few of the areas from complete destructive lesions (traumatic) of the cord or of the roots. Thorburn gives an exceedingly instructive case of a lesion of the cord at the level of the first dorsal root. The intrinsic muscles of the hands, which we know are supplied from the first dorsal root, were paralysed. The anæsthesia was limited "with the greatest accuracy" to the side of the arm and fore-arm being bounded in front by a line running vertically from the front of the deltoid opposite the second rib down the centre of the triceps and forearm to the middle of the wrist; similarly behind the boundary was a vertical line down the centre of the triceps and back of the forearm to the centre of the wrist. In the hand the limiting line sloped both in front and behind from the centre of the wrist to the cleft between the fourth and fifth fingers, the little finger being completely anæsthetic, and others not so. The trunk was anæsthetic as high as the level of the second rib in front and the spine of the seventh cervical vertebra behind. Compare the upper line with that in plate I. and II., or with the upper line found by placing together the three herpetic eruptions shown in fig. 12, 13 and 14, and they coincide in a remarkable manner. Now the words limited "with the greatest accuracy" makes me think that the limit given for the "anæsthesia" was in reality the limit of analgesia. For so accurate an observer as Thorburn would never have described the vague line of anæsthesia to touch as "accurately limited." Moreover I cannot but think that if touch, pain, and heat and cold have all been tested the fact would have been mentioned.

Thus I think we can assume that the area marked out on plate I. and II. by D 1 represents a disturbance in the course of the first dorsal. Having determined that the area marked D 1 on plate I. and II. represents the supply of the segment of the spinal cord corresponding to the first dorsal root we can now count downwards, and number each area as has been done on the coloured plates.

How far does this enumeration correspond to what we know of other cases of lesion of the cord.

On p. 69 Thorburn gives a case of fracture dislocation of the sixth dorsal vertebra. The anæsthesia extended on the trunk "as high as the eighth rib." This description corresponds very fairly with the upper limit of the subscapulo ensiform pain area [plate I. and II., D 7.] The cord was completely cut across at the level of the sixth dorsal vertebra. Now the sixth dorsal root arises from the cord at the lower border of the fifth dorsal vertebra and the seventh root was the highest whose origin was completely destroyed. From our calculation we concluded that the subscapulo-ensiform must be supplied from the segment of the cord corresponding to the seventh dorsal root.

Again fig. 17 shows the upper limit of analgesia, *i.e.*, loss of appreciation of the prick of a pin in a traumatic spinal case. Mr. Horsley operated and found that owing to a fracture of the first and second lumbar vertebræ the roots at the level of the body of the twelfth dorsal vertebra were firmly matted up and apparently destroyed. The cord itself was apparently uninjured and pulsated down to the end. Moreover the area supplied by the lower sacral roots was unaffected. The reaction to faradism was completely lost in the muscles of the legs, calf, and back of thigh. Thus we must assume that the roots involved in this tissue were degenerated. The highest root which comes off at the level of the twelfth dorsal vertebra is the first lumbar. Now the upper limit of the analgesia corresponds very fairly with the lower limit of the sacro-femoral area [*vide* plate I. and II., D 12], which by enumeration from the first dorsal was assumed to correspond to the distribution of the twelfth dorsal root segment.

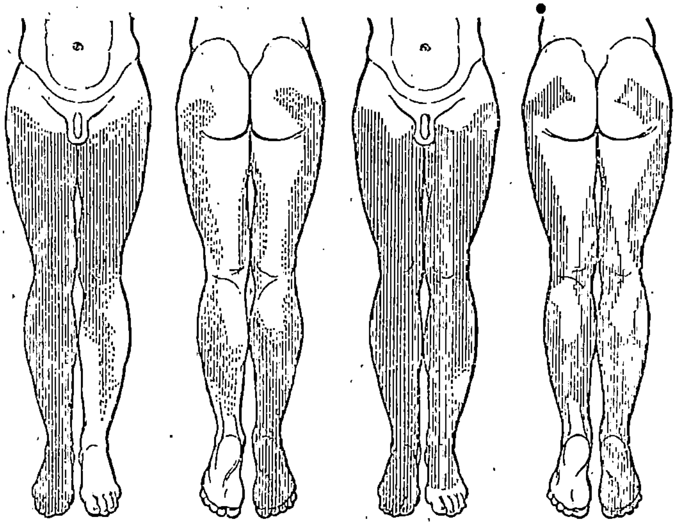

FIG. 17.

To show the disturbance of sensation in Case No. 19.

The left half of the figure shows the areas over which the sensation of touch was lost. Note the ill-defined edges.

The right half shows the areas over which the sensation of pain, heat and cold were lost. The edges are better defined; but are not absolutely sharp, probably owing to the fact that the loss of sensation was due more to affection of the roots than of the spinal cord.

Case 19.—James S., aged 33, fireman. U. C. H. (Mr. Horsley), November, 1891.

On night of April 14, 1891, went to bed drunk and walked out of the window 40 ft. from the ground. Was unconscious for three to four weeks.

Condition on admission.—Marked kyphosis in dorso-lumbar region. Whole dorsal region arched and spine rigid. Summit of kyphotic curve formed by the exceedingly prominent spine of the twelfth dorsal vertebra which stands out fully three-quarters of an inch beyond the rest. This prominent spine has a bifid extremity and feels almost as if it were two spines side by side. No tenderness on firm pressure.

Lower limbs rigid and nearly motionless. The feet are in the position of talipes equino-varus. Feet and ankle joints are stiff. In the knee joint passive flexion is possible up to 30° without undue force.

Complete loss of power in all the muscles of the right lower limb. No independent action in psoas and iliacus, but he is able to raise his thigh from the bed by lifting up the pelvis. On the left side he can voluntarily contract the following muscles, rectus, vastus externus, and to a less extent vastus internus, abductor muscles including adductor magnus. No action in hamstrings or glutei on either side.

Muscles on front of thigh and tibialis anticus react on both sides to faradism. On the left side the hamstrings, calf muscles and flexor brevis digitorum react, but on the right side these muscles do not react to faradism.

Muscles on front of thigh and tibialis anticus react to galvanism on both sides. The muscles of the calf and hamstrings also react on the left side but not on the right. A stronger current is required on the right side than on his left. A. C. C. appears early but does not preceed K. C. C.

Sensation of touch lost over the areas in fig. 17 left half. Penis and scrotum unaffected.

Sensation of pain lost over the areas in fig. 17 right half. Penis and scrotum unaffected.

Sensation of heat and cold follows the same lines as those of pain (fig. 17 right half.)

Knee-jerk abolished both sides. Right plantar reflex abolished but left is present. Cremasteric reflex abolished on both sides. No ankle clonus. Abdominal reflexes normal. No affection of upper limbs, trunk or head. Discs and fundus oculi normal.

Urine dribbles away. Ammoniacal and contains much mucus. He suffered from cystitis for several months before admission. Has control over the rectum and sphincter ani. Bowels open every two or three days.

December 12, 1891.—Mr. Horsley operated. He removed the arches of the eleventh and twelfth dorsal, and first and second lumbar vertebra. Across the lamina of the second lumbar on the right side was a groove formed by a fracture. Lamina itself was tilted so that its upper edge was directed forwards as well as upwards in a plane about 45° to the frontal plane. Opposite the eleventh dorsal vertebræ the cord was normal and pulsated with respiration. Opposite the twelfth dorsal the theca vertebralis was much thickened of a yellowish white colour and pulsation was just visible. Opposite first and second lumbar colour and general appearance was normal and pulsation was distinct. Over the twelfth dorsal vertebra the adhesion of the theca to the bone was so close that it was impossible to pass a probe between the theca and the anterior part of the vertebra. Theca was opened. Opposite the twelfth dorsal vertebra it was adherent to the posterior surface of the cord and also to the nerve roots. They were so thickly matted that it was quite impossible to disengage them. Made an uninterrupted recovery from the operation.

Therefore I think we can say that the area which runs down the ulnar side of the arm corresponds to the supply of the first dorsal segment, whilst the area which forms a flap on the outer side of the thigh and crosses the thigh to reach the inner side above the knee corresponds to that of the first lumbar root.

But now we reach the gap and can count no further from above downwards.

Let us then proceed to the consideration of the areas on the back of the thigh on the sacral side of this gap. The tract of skin figured on page 82, which was affected in consequence of a lesion to the mucous membrane of the bladder closely resembles the area of analgesia in a case of destruction of the spinal cord at the level of the third and fourth sacral roots reported by Oppenheim.[1] Now I pointed out above that I believe this area is made up of two portions, and I think we can now assume that the saddle-shaped area on the buttock, with its maximum tender point at the ischial tuberosity represents the supply of pain fibres from the segment corresponding to the third sacral root, whilst the one above it, with a well marked tender point over the lower sacral spines, corresponds to the fourth sacral.

If this be so, the long thin area on the back of the thigh will represent the supply of the second sacral segment, whilst the soleal area will correspond to the distribution of the first sacral segment. Thus if the spinal cord at the level of the first, second, third and fourth sacral roots were destroyed, we should expect to get loss of sensation to pain over an area made up of these four tracts.

Now Herter and Starr[2] report a case in which this lesion was present, and the resulting analgesia bears a fairly close resemblance to a combination of the four areas marked out by tenderness in visceral disease.

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[1] *Archiv. f. Psychiat.*, xx. 298.

[2] *N. Y. Med. Journal*, Aug. 9th.

Now the next area above the soleal which appears in visceral disease occupies the outer side of the leg and swings downwards to cover the dorsum of the foot as far forwards as an inch from the root of the toes. Here it borders on the soleal area. If, therefore, our enumeration is correct, this tract of skin must represent the supply of the pain fibres of the fifth lumbar segment.

Here, again, we come to the gap or tract of skin which is not affected in visceral disease. Thus this gap is now seen to be bounded above by the lower border of the first lumbar area and below by the upper border of the region supplied by the segment of the cord corresponding to the fifth lumbar root. Here, then, our areas of tenderness are no help in the delineation of the roots.

Fig. 18 shows the extent to which pain, heat and cold were lost in a case under the care of Dr. Hughlings Jackson, which he kindly allows me to mention. The patient was a man aged 27, who nearly two years before admission to the National Hospital, Queen Square, had fallen from a scaffold. In the right leg we find that the long patch so frequently seen in lesions about this region, extending down the inner side of the leg to the root of the metatarsal of the great toe is unaffected; whilst on the left side it is analgesic. Now Starr associates this patch with the third lumbar root. My material at present is too scanty to decide this point, but I have so frequently found that this area is affected when the areas about the knee are unaffected, and *vice versa*, that I feel inclined to think that it forms a separate root area of the form I have shown in fig. 20. It extends from the anterior border of the tibia inwards and backwards to the middle of the calf. Downwards it reaches the base of the metatarsal bone of the great toe and above it is forked, to receive a downward process of the area above. Now as this area comes next above that already described as the fifth lumbar, it probably represents the distribution of the pain fibres of the fourth lumbar root.

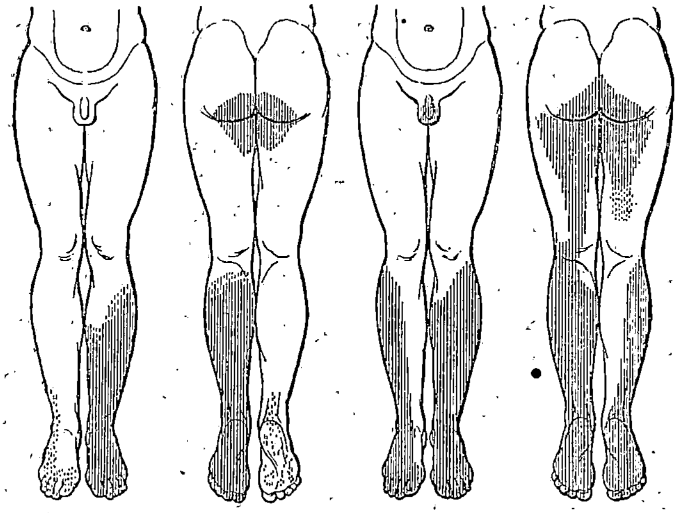

FIG. 18.

To show the disturbance of sensation in Case No. 20.
The left half of the figure shows the loss of sensation to touch.
The right half of the figure shows the loss of sensation to pain, heat and cold.
Note that the loss on the right half is more extensive and better defined than that on the left half of the figure.

Case 20.—Edwin C., aged 27, labourer. Queen Square (Dr. Hughlings Jackson), May, 1892.

August 8, 1890.—Fell from a scaffold. Was not at first unconscious, but became so afterwards.

Condition on admission.—Intelligent, healthy-looking man. Over lower end of sacrum and coccyx is a large irregular scar, with a small superficial area of ulceration. This is a bedsore, which has evidently at one time been much larger than it is now.

His motion, sensation and reflexes above the level of the pelvis are normal. Cranial nerves and special senses normal.

Can flex right hip well. Flexion of left hip present, but feeble. Extension of both hips feeble, but better on right than on left side.

Can extend right knee well. Extension of left knee considerably less than right. Cannot flex either knee when lying on his back. When lying on his face he can raise the right foot off the bed and flex the right knee to a right angle. The left foot can only just be lifted off the bed in this position. This action appears to be due almost entirely to the semi-membranosus.

Absolute paralysis of both feet, and all the toes. Both feet, especially the left, are in a position of simple talipes equinus without contracture. No rigidity.

Considerable wasting of both legs below the knees, and of the flexor and extensor aspect of the thighs. All the wasted muscles are flabby and soft.

Electrical reactions.—

	Faradism.		*Galvanism.*	
	Right.	Left.	Right.	Left.
Quadriceps	good	strong current	A.C.C > K.C.C.	0
Hamstrings	0	0	0	0
Glutei	?	?	0	0
Tibial Group	0	0	0	0
Peroneal Group	0	0	0	0
Sural Group	0	0	0	0

Tactile Sensation.—Lost over the areas in fig. 18 left half. The sensation of the penis and scrotum are perfect.

Painful Sensation.—Lost over the areas in fig. 18 right half. Complete loss of painful sensation in penis and scrotum, excepting over a small area at the base of the scrotum.

Sensation of heat and cold accurately follow the lines of painful sensation (fig. 18 right half).

Localises well over the areas where sensation is present. No allocheiria.

Reflexes.— Knee-jerk slight on right side. Absent on left side. No ankle clonus. Plantar reflexes not obtained. Abdominal reflexes and all upper reflexes, well obtained.

He knows when he wants to pass water, but has no power of retaining it. He can feel the passage of urine when it reaches the orifice. Urine 1024, faintly acid, trace of albumen. No sugar.

Feels when his bowels require to act, but is not conscious of the actual passage of fæces. Has no power of retarding action.

Complete loss of sexual desire since the accident. No involuntary erections. Occasional emissions without erections.

The next area above this is extremely difficult to describe in words, and its limits are better grasped from an examination of fig. 20. A case of herpes zoster shown in fig. 19 occupies this area in addition to the areas which we have already learnt to associate with the fourth and fifth lumbar. The characteristics of this area are its long upward flap on the inner side of the thigh over the adductors, and its distribution to the area around and over the patella. It has a small downward flap on the inner side of the leg behind the knee which fits into the fork of the area below (fourth lumbar).

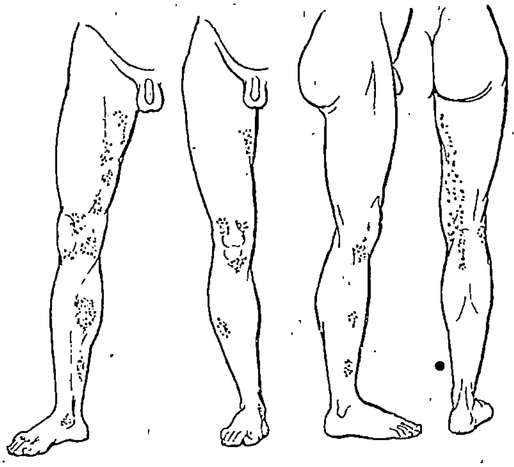

FIG. 19.

To show the distribution of the herpetic eruption in Case No. 21. It is supposed to occupy mainly the areas belonging to the 3rd and 4th lumbar segments with slighter implication of the 5th lumbar.

Case 21.—Henry M., aged 22, June 28.

Last week he had a severe cold. He had a sore throat and a "whistling" cough.

Eruption came out June 19. No antecedent pain.

No tenderness except over the areas of the eruption.

Appetite poor. Bowels open regularly. No gonorrhœa or syphilis.

Thorburn (p. 132) gives a case of spinal injury where the upper limit of the anæsthesia was two inches above the knee joint. It was "concave downwards," and lower on the inner than on the outer side of the knees. This anæsthesia was caused by transverse divisions of the cord at the level of the origin of either the third or the fourth lumbar roots. As he expressly mentions that the posterior aspect of the leg was not examined when the anæsthesia was thus limited it is possible that this is the knee portion of the area I have just described.

This complicated area represents so far as I have yet seen the distribution of the pain nerves from that segment of the spinal cord corresponding to the third lumbar root.

Now if we place together on a figure all the areas we have determined so far, in the gap between the first and fifth lumbar roots, one only remains, having the following distribution. Its most posterior portion is situated over the buttock and does not quite reach the middle line behind.

It fills in the diamond shaped gap between the posterior part of the first lumbar and the upper part of the third and fourth sacral areas. Thence it sends a downward flap, whose posterior border corresponds to about a line drawn from the posterior border of the trochanter to the inner edge of the biceps at the knee. The anterior border of this flap is bounded by the posterior border of the descending flap of the first lumbar root area. On the front of the thigh it occupies the vacant space over the quadriceps, between the inner and the outer flap of the

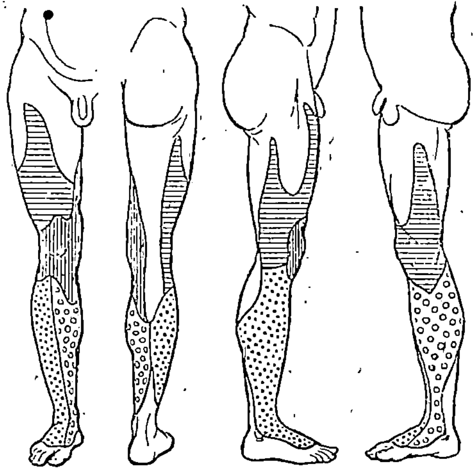

Fig. 20.

To show the presumed distribution of the areas supplied from the 2nd to the 5th lumbar segments. Of these the 2nd, 3rd and 4th lumbar comprise the "gap" which is not normally affected in disturbances of the the viscera.
The area marked in *cross lines* represents L 2.
The area marked in *vertical lines* represents L 3.
The area marked with *circles* represents L 4.
The area marked with *dots* represents L 5.

first lumbar area and ends just above the knee, and on the inner side by adjoining the ascending limit of the third lumbar.

Thus we have now filled up the gap in the leg which is unaffected in visceral disease, and seen that it probably represents the supply of pain fibres from three segments, the second, third and fourth lumbar.

It would take me too long to discuss here how the gap on the arm is to be filled up, and I must reserve the full discussion for a future paper on the areas of the head and neck.

Now the descending branches of the cervical plexus which came off as low as the fourth cervical root supply the skin over the first two ribs and over the point of the shoulder. This represents the lowest limit of the areas of referred tenderness in the head and neck.

Thus the gap in the arm which is unaffected by visceral disease probably represents the distribution of the pain fibres of the fifth, sixth, seventh and eighth cervical segments.

Conclusions.

We have now been able, with the help of traumatic lesions of the spinal cord, to name each area according to the spinal segment to which it belongs. Thus the dorso-ulnar area represents the supply of the first dorsal segment, and the gluteo-crural that of the first lumbar, and each of the eleven intervening areas represents the supply of one segment whose number we now know.

In a similar manner we found that the gluteo-pudendal area represented the supply of the third sacral segment—the fibulo-dorsal that of the fifth lumbar, and the intervening areas have been numbered according to the segments to which they belong

We have also seen that the "gap" corresponds to the second, third and fourth lumbar segments, and we have been able from the lesions produced by organic disease to approximately apportion the skin into areas corresponding to each of these segments.

CHAPTER III.

PAIN AND TENDERNESS IN DISTURBANCES OF SPECIAL ORGANS.

Introductory.

We have now seen that the whole of the body and limbs can be marked out into areas, each of which I have attempted to show, corresponds to the cutaneous distribution of the pain fibres given off from one segment of the cord. I shall now try and put before you the extent to which these areas are affected in diseases of the various organs.

It is a well-known fact that the pain of visceral disease is not always localised by the patient over the organ affected. Ross first explained this fact by the ingenious hypothesis that the pain of visceral disease was referred along the distribution of the somatic roots which came off from the same level of the cord as the sympathetic fibres to the organs affected. He collected a large number of cases of pain, and his intimate knowledge of the sensory distribution of certain roots enabled him to say definitely that in certain cases, the distribution of the pain corresponded exactly with that of certain roots.

The description given by patients of their pain is, however, so unsatisfactory, that this important work did not bear fruit to the extent that might have been expected.

An enormous advance was made when Dr. Mackenzie[1] described the cutaneous tenderness which is so frequently associated with the pain of visceral disease. For here we have a symptom which is distinctly more objective than that of pain only. He proceeded to collect a large number of cases in which the tenderness was present over one or more points, and in the course of this section, I shall repeatedly refer to his interesting paper. Although we worked independently and published at the same time, our results agree to an extent that is very flattering to me. In particular, I wish to emphasise the following points in which we are at one.

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[1] *Medical Chronicle*, August, 1892.

1. The pain of which the patient complains always lies within the areas of tenderness, if these be present. Should they be present at one time and absent at another, but the pain remain, tenderness is always found to make its appearance in the area which is said to be painful.

2. The tenderness is not deep seated, but is purely cutaneous or subcutaneous. One of the best methods of eliciting it, is to pick up the skin gently between the finger and thumb. In addition, Mackenzie uses the point of a pin, but I generally use the broad blunt head of a large pin, especially where the area of skin affected lies over a bony substratum. Thus Mackenzie looks for the exaggerated expression of pain produced by the point of the pin over the tender area: whilst my method of stimulation produces no pain until the tender area is reached.

3. These areas of tenderness bear a definite relation to the different organs affected, but in many cases lie at a considerable distance from the organ affected. The pain produced by stimulation is not produced by any action on the organ itself, which may lie at a considerable distance from the tender spot. Again, the tenderness may be on the right side, though the organ affected lies on the left side of the body.

4. The superficial reflexes are usually exaggerated over the tender areas produced by visceral disease.

I shall now enumerate very shortly the areas which are affected in diseases of the various organs, and shall now use the segmental nomenclature I determined in the last section instead of the cumbrous names for the areas which were necessary before we had determined their significance.

The Heart and Aorta.

In diseases of the heart and great vessels, it is easy to get an account of the pain, but it is often difficult to demonstrate tenderness; for notoriously the most painful disturbances take the form of anginal attacks, in which the patient is not in a fit state to give an account of himself. Sometimes, as is well known, the pain radiates down the ulnar side of the arm to the wrist or little finger, and sometimes it starts from this point and radiates up to the chest.

The first case I have selected for illustration, is that of a man, aged 40, who had suffered from attacks of pain three to four times a week. The heart was dilated and hypertrophied, and there was a double aortic and faint mitral systolic murmur. He first of all feels as if he were blown up with wind, then pain comes on under the left breast, and at the angle of the left scapula. It then appears at a point in the third space, just internal to the nipple line. In these attacks, a spot in the second space 3 inches from the middle line, and thence down the arm to the fingers, but not to the thumb. In the arm it darts backwards and forwards, sometimes upwards, sometimes downwards. I subsequently saw him in one of these attacks, and he then had intense cutaneous tenderness over the ulnar side of the forearm, back and front, over an area in the supra-spinous fossa at the level of

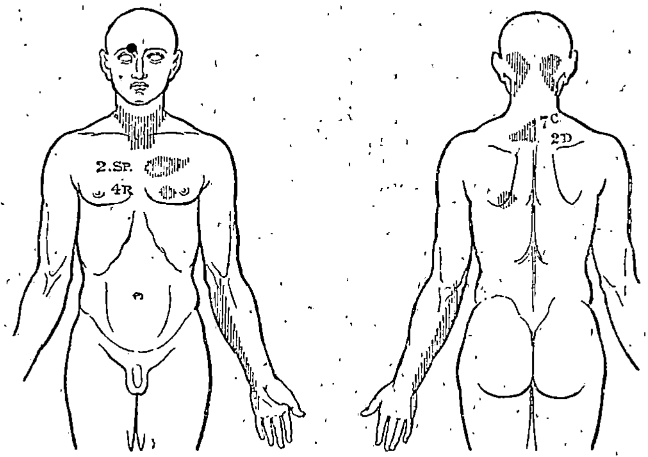

FIG. 21.

To show the areas of cutaneous tenderness during an anginal attack in a man with aortic disease (Case No. 22). They represent the whole of the 1st dorsal and 3rd cervical segments together with the maxima of the 4th dorsal.

2 Sp. is placed over the 2nd intercostal space.
4 R is placed over the 4th rib.
7 C at the level of the spine of the 7th cervical vertebra.
2 D at the level of the 2nd dorsal spine.

the second dorsal vertebra, and over an area in the second space extending from the nipple line to the mid-line of the chest. Thus the tenderness marked out very closely the area supplied by the first dorsal segment. In addition, he had pain and tenderness over the angle of the scapula, and over a point on to the fourth rib just internal to the nipple.

Case 22.—J. C., aged 40, draper's porter. Was in University College Hospital a few months before I saw him first with heart disease. The heart is now dilated and hypertrophied. Cardiac impulse strong. No thrill. Double aortic and mitral systolic murmur. Arterial pulsation up to ear. Capillary pulsation marked. Pulse 84, typically aortic regurgitant.

Suffers from attacks of pain as often sometimes as three and four in the week. He first feels blown up with wind, then pain comes on under left breast, and at angle of scapula. It then goes to a point just above the nipple; thence to a point in the second space from which it radiates down the arm to the hand. The ulnar side of the arm only is affected. After the pain in the arm comes on, he gets pain in the throat, with hoarseness and difficulty in speaking. In about an hour he vomits and brings up wind, and the attack is at an end.

On June 22, he had cutaneous tenderness (1) over the fifth rib internal to the nipple on left side. (2) Over the vertebral border of the spine of the scapula. (3) Over a point just internal to the angle of the scapula. (4) Over the ulnar side of the bend of the forearm. (5) Over the base of the hypothenar eminence in the left hand.

June 29. Came to U. C. H. in evening with a bad anginal attack. Attack began at 10.30 p.m. as described above. At midnight he was in great pain, face anxious and pale; great cardiac distress. Pulse could not be counted; was running and of very low tension.

There was intense cutaneous tenderness as in fig. 21, and the pain was referred entirely to the regions over which the skin was tender.

Voice hoarse. No cough. Pupils equal, about 5 mm. React normally.

He was put to bed and soon began to vomit. By 1 a.m., June 30, the attack was over.

At 8 a.m., June 30, he was well, and all cutaneous tenderness had practically gone. He left the Hospital to go to his work.

In the case of a man, aged fifty, who suffered from an aortic aneurism pointing to the region of the second rib, the tenderness was situated over the area shown in fig. 22, corresponding to the second, third, and fourth dorsal segments.

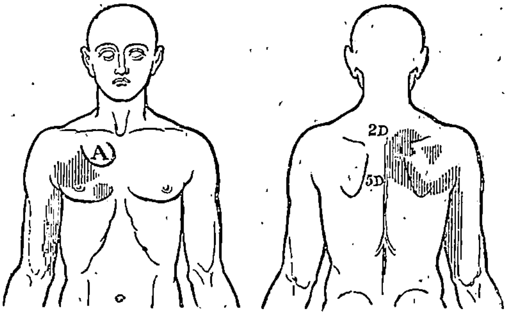

FIG. 22.

To show the areas of cutaneous tenderness in a man with aortic aneurism (Case 23). They represent the 2nd, 3rd and 4th dorsal segmental areas.
The outlined area A represents the extent of visible pulsation.
2 D is placed at the level of the spine of the 2nd dorsal vertebra.
5 D at the level of the spine of the 5th dorsal vertebra.

Case 23.—Edward T., aged 50, caretaker. U. C. H. [Dr. Roberts.]

November, 1890. Attack of gout. Pain in right side of chest and shortness of breath. By June, 1891, this pain had so increased that he could not walk without becoming short of breath and suffering from pain. July 15, U. C. H. Double aortic murmur, aortic pulse, aneurism of ascending and transverse portions of aorta diagnosed.

June, 1892, re-admitted. A little to right of middle line in first and second spaces is a swelling extending from the lower border of the clavicle to the third rib below. To the left it reaches the middle line of sternum; to right, nipple line. Surface of the pulsating swelling fairly regular except that there is a nipple-like projection in the centre.

Apex beat, fifth space half-inch outside nipple line. Diastolic aortic and less marked systolic aortic murmur over the swelling and over sternum.

Pulse 76, aortic. Pupils equal. No anginal attacks.

Pain on right side of chest:—

(1) in third space about 2½ inches from mid-sternum.

(2) Pain at extreme apex of axilla.

(3) And over a point behind at the level of the spine of the scapula a little to right of middle line.

July 3. Whilst singing hymns sudden increase of the pain and extension of the area of the aneurism; very marked cutaneous tenderness as in fig. 22.

Another case of dilatation of the aorta with dilated and hypertrophied heart showed the second dorsal segmental area in a very beautiful way. The maximum pain was situated over the most posterior portion of the spine of the scapula and over the second space in front. This pain radiated down the arm, but did not go further than the elbow.

Mackenzie[1] gives a number of cases of cardiac disease showing pain and tenderness over these areas, and gives an excellent figure showing the whole area affected in such cases. He points out that in a large number of cases certain tender points make their appearance. A glance at plates I. and II. shows the segments to which they belong, according to my enumeration, but I will group these points according to the segmental areas of which they are the expression.

First Dorsal.—	(*a*)	Seventh cervical to second dorsal spine.
	(*b*)	Second space from nipple line to mid-sternum.
	(*c*)	Ulnar side of bend of elbow.
Second Dorsal.—	(*a*)	Over the spine and vertebral border of the scapula.
	(*b*)	Over the axillary border of the pectoralis major.
	(*c*)	Over the middle of the triceps muscle in the arm.
Third Dorsal.—	(*a*)	Over the middle of the scapula just below the spine.
	(*b*)	Over the third rib internal to the nipple line.
Fourth Dorsal.—	(*a*)	Over the third and fourth vertebral spines and outwards to the vertebral border of the scapula.
	(*b*)	Over the region in the axilla covered by hair.
	(*c*)	Just above the nipple itself.
Fifth Dorsal.—	(*a*)	Exactly over the angle of the scapula.
	(*b*)	Over the fourth rib and space internal to the nipple.

In cases of angina pectoris, whether there are physical signs of valvular disease or not, we frequently find that the

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[1] *Loc. cit.*, fig. 4.

areas corresponding to the sixth and seventh dorsal roots are also affected. But in addition other areas belonging to the cervical plexus are also well marked. I shall therefore leave the full consideration of the phenomena of anginal attacks for my next paper on the head and neck. For in order to understand their full significance we must first know the distribution of the segmental areas of the cervical plexus.

Thus to sum up the results yielded by a study of cardiac disease :—

(1) In diseases of the heart, and more especially in aortic disease, the pain is referred along the first, second, third and fourth dorsal areas.

(2) In angina pectoris the pain may be referred in addition along the fifth, sixth, seventh and even the eighth and ninth dorsal areas, and is always accompanied by pain over certain cervical areas.

Lungs.

For the production of visceral pain, and especially of tenderness in connection with the pain the disturbance in the organ has to be fairly acute. Now with regard to the lung we labour under several disadvantages if we attempt to find tenderness associated with its disturbances. For the majority of cases which occur in our hospitals are chronic,

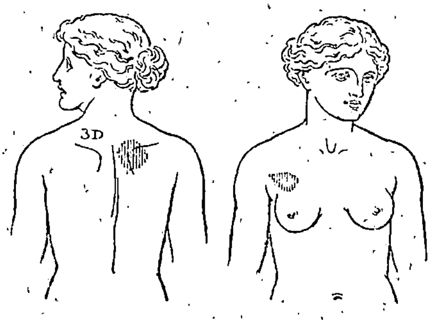

FIG. 23.

To show the areas of cutaneous tenderness in a case of acute bronchitis (Case No. 24).

These spots represent the maxima of the 2nd dorsal segment.

and those which are acute, such as lobar pneumonia, are not as a rule in a condition to give an account of themselves.

Fig. 23, however, shows the position of intense pain in a case of acute bronchitis. The pain was particularly bad on coughing. The areas in this figure also show the position of great cutaneous tenderness. The patient said she was so sore "she felt as if she had been beaten." The signs of bronchitis were far more marked on the right side than the left in this case.

Case 24.—Mrs. B., aged 34.

Pregnant seven months. Called to her on account of bad cough and pain in chest, which came on four days before. Temp. 103. Pulse 110. Respn. 30. Numerous mucous râles all over right side and also at left base. No dulness. Pain on coughing, associated with great cutaneous tenderness over the areas in fig. 23. Pressure over these areas, especially over the one in the back, seems to start the cough.

She subsequently aborted, and died June 8. No definite signs of pneumonia. Lochia normal. No pain or disturbance connected with uterus or other organs of the body.

With regard to phthisis which is the lung disease on which one has to depend, an enormous number of cases have neither pain nor tenderness. Some have pain and only a few have definite cutaneous tenderness. These as far as I have seen so far are those with an irritable cough in the early stage. Many will tell you they have suffered pain "but that has passed off now." Perhaps the commonest place is over the same areas as those in fig. 22. Another very common area to be affected is that of the sixth dorsal segment. The two main points of this area are situated the one posteriorly at the level of about the seventh dorsal spine just internal to the angle of the scapula and the other in front under the breast. The lowest area or the lowest two areas of the cervical plexus are also frequently affected. To these areas belong the tender points at the posterior aspect of the shoulder joint, and those over the second rib and under the clavicle. The inner side of the arm and the axilla are also sometimes affected, and not uncommonly the pain and even tenderness may extend down the ulnar border of the arm and forearm to the ulnar side of the hand.

The case mentioned on p. 34 shows the possible connection between the area of these referred pains and the area occupied by herpetic eruptions. A man of 34 years of age, with evident, though slight, signs of advancing phthisis at the right apex was accustomed to have pain and a sore feeling over the third dorsal spine and the spine of the scapula "whenever he caught cold" (*i.e.*, whenever his cough was worse). He came to me "having caught a fresh cold," and this time the pain to which he was well accustomed did not pass off but was followed by a herpetic eruption over the scapula and in the axilla corresponding to the area of the third dorsal segment.

Thus in diseases of the lungs the areas corresponding to the first, second, third, fourth, fifth, sixth and seventh dorsal segments may be affected. The second, third, fourth and fifth dorsal seem to be most commonly affected. There may also be pain and tenderness over the lower areas such as that of the seventh dorsal but in such cases there is frequently distinct evidence of dyspepsia, so common in the early stages of phthisis.

Œsophagus.

Stricture of the œsophagus affords one of the most definite instances of referred pain for here the pain can be produced by swallowing. In several cases the necessity for passing a bougie has been taken advantage of to discover the situation of the pain. As the bougie passes the patient is asked to mark out with a pencil the spots where the pain is situated. In most cases the pain is referred to a point about two inches from the middle line exactly at the angle of the scapula and is associated with a pain in front over the fifth space about half-inch internal to the nipple line. There is sometimes pain in the back over the sixth and seventh vertebral spines. Frequently pain thus referred is the only phenomenon but sometimes cutaneous tenderness is also present over the spots of referred pain.

One of the most marked cases I have seen was in a man of 65 years of age who in consequence of diet and treatment presented scarcely any pain. The stricture, which was fourteen inches from the mouth, began to close and it was therefore determined to pass a bougie. After the passage of the instrument he complained of a good deal of pain and I found the whole of the fifth, sixth, seventh and eighth dorsal areas exquisitely tender. The curious upper border of the fifth dorsal area with its axillary flap was beautifully shown. By the next day this tenderness had subsided and was replaced by tender points only. These tender points in turn subsided one by one. He died a month afterwards of malignant disease of the œsophagus.

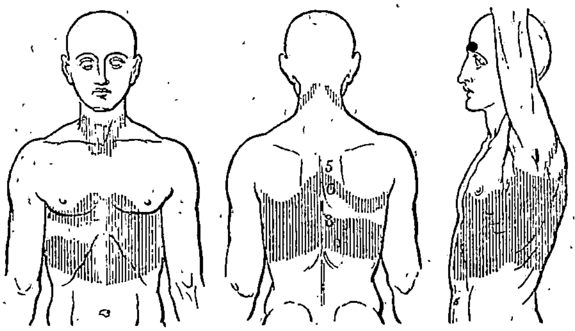

FIG. 24.

To show the areas of cutaneous tenderness in a case of stricture of the œsophagus, induced by the passage of a bougie (Case No. 25).

The areas represent the supply of the 5th, 6th, 7th, and 8th dorsal segments on the left side, and the 5th, 7th, and 8th dorsal on the right side. The upper border of the 5th dorsal is well shown on the lateral figure.

The numbers on the posterior view represent the spines of the respective dorsal vertebræ.

Case 25.—Samuel L., aged 64. (Mr. Heath, U.C.H.)

One month before admission difficulty in swallowing solids. Gradually getting worse. Now even liquids regurgitate. Healthy looking man. Somewhat wasted. When he attempts to swallow milk he makes efforts of deglutition for about 15 seconds and then milk comes up again.

Complains of pain at episternal notch and over a point in the fifth space just inside the nipple on both sides. Also sometimes in the back just internal to the angle of the scapula. Cutaneous tenderness more or less marked over all these points.

No physical signs of aneurism. Voice normal.

No physical signs in lungs or heart.

No enlarged glands. Liver not enlarged.

Took prepared foods well and became almost free from pain and cutaneous tenderness. June 8, an œsophageal bougie was passed

14 inches from teeth but it was found impossible to get it into the stomach. Tenderness the same evening as in fig. 24. Very similar results obtained on June 15 after similar abortive attempts to pass bougie.

Died in August of complete closure of maligant stricture.

As the bougie passes the patient frequently complains of "choking pains" at the episternal notch. I have occasionally, though rarely, found tenderness associated with this pain, marking out one of the lower cervical areas.

Thus the most prominent situation of pain and tenderness in stricture of the œsophagus corresponds to the distribution of the fifth dorsal, but the sixth, seventh and even the eighth dorsal segments are also frequently affected more or less.

The Breast.

Many factors combine to hinder the determination of the referred pain and tenderness due to disturbances of the breast. For it is notorious that the majority of tumours of the breast give no referred pain, and secondly, whenever a woman is run down, from whatever cause, she tends to suffer from a pain under the breast, associated with tenderness; moreover, the disturbance has to be fairly acute, or reference does not take place.

The most perfect example of referred pain in its purest form, was produced by retention of milk in one breast. Here there was marked cutaneous tenderness over the fourth and fifth dorsal roots. Fig. 25 was taken from a

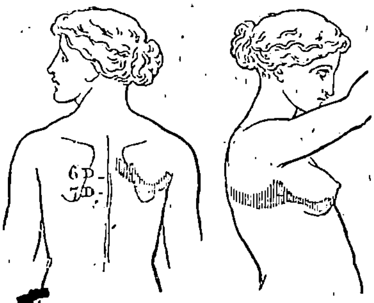

FIG. 25.

To show the distribution of cutaneous tenderness in Case No. 26. This area represents the distribution of the 5th dorsal segment.

young woman whose child was 4 months old. She suffered from sore nipples, and every time the child sucked she had pain over the angles of the scapulæ, over the posterior part of the axilla, and over the lower half of the breast. The tender area corresponded exactly with that of the fifth dorsal root.

Case 26.—Mrs. B., aged 34. Is suckling 4 months' old child. Right nipple was sore, and bled when the child sucked. This is now healed, but she complains of pain in the breast and pain in the back, between the posterior border of the scapula and the spine, on the right side, at about the level of the sixth or seventh dorsal spine. This pain is accentuated every time the child sucks. Marked cutaneous tenderness, as in fig. 25. No mastitis. Temperature normal.

It is not at all an uncommon thing for women who suckle children into the second year to complain of pain "over the tip of the shoulder blade." Mostly such women are debilitated and weakly, but I made the following observation in a strong healthy woman who was suckling an 18 months' child to prevent conception. She was free of pain or tenderness before she put the child to the breast, but after the child had pulled violently and vigorously at her breast for a few minutes, the pain over the shoulder blade began to appear. She let the child suck until he fell asleep, and by that time there was a distinct area of cutaneous tenderness over the lower border of the scapula. She was accustomed to let the child pull at the breast whenever he woke in the night, and she said that in the morning her shoulder blade was sometimes so sore she could not put on her stays.

Thus the principal pains and tenderness in disturbances of the breast gland and ducts (other than new growth) seems to be situated over the fourth and fifth dorsal roots.

Stomach.

Pain in gastric disturbances is almost always accompanied by some cutaneous tenderness. This tenderness lies in front between the level of the ensiform cartilage and the umbilicus. It occupies one or more of the three areas corresponding to the supply of the seventh, eighth and ninth dorsal segments.

One of the commonest situations for pain is at the epigastrium, just over the ensiform cartilage, and a reference to plate IA, shows that this is one of the maximum spots of the sub-scapulo-ensiform, or seventh dorsal area. In cases of so-called gastric ulcer, cutaneous tenderness is nearly always present over this area and over a point in the line of the angle of the scapula at the level of the ninth dorsal spine.

The middle area of this group (eighth dorsal) also appears frequently, but as far as my experience goes, seldom alone in stomach affections. It is nearly always associated with the one above or the one below it. Its characteristic tender point is situated over the eighth space, almost exactly in the mid-axillary line.

The next area, corresponding to the ninth dorsal root, is one of the commonest of all the stomach areas. It appears repeatedly in cases of gastric ulcer, either alone or in combination with one or more areas of the group.

It is a curious thing that all these areas associated with the stomach tend to appear bilaterally. This was so in a case of which fig. 26 was taken. It represents the cutaneous tenderness which appeared in a youth who was suffering from a violent attack of stomach pain, accompanied by vomiting, as a consequence of irritant food.

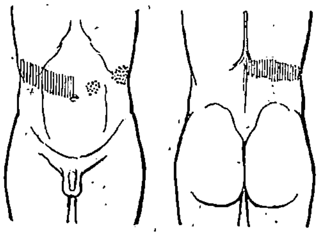

FIG. 26.

To show the distribution of cutaneous tenderness in a case of acute gastritis (Case No. 27). The area on the right side represents the full extent of the 9th dorsal area, whilst the spot over the costal border on the left side represents its most prominent maximum.

Case 27.—Youth, aged 14. Pain in the abdomen for the last twelve hours. Intense—keeps him doubled up. Points out position of the pain by placing the flat of his hand just above the umbilicus, with the fingers pointing to the linear alba. Cutaneous tenderness (blunt end of pin called "prick") over the area in fig. 26.

Bowels open. No constipation or diarrhœa. Has vomited several times.

Pain and tenderness gone next day.

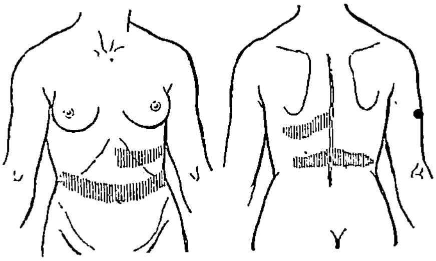

FIG. 27.

To show the distribution of cutaneous tenderness in a case of so called gastric ulcer (Case No. 28).

The areas on the left side represent the distribution of the 7th and 9th dorsal segments. That on the right side represents the distribution of the 9th dorsal segment.

Case 28.—M. E., aged 27, single, servant.

Two years ago attack of pain and vomiting. No blood. Recovered almost completely, but was subject to pain after food.

November 4, 1891, felt sick, and at 8.30 a.m., vomited large quantity of altered blood. Next day passed half pint of black stuff by bowel.

Constant cutaneous tenderness, mostly situated over the upper area; increased by food or by anything warm, but never quite absent. Pain referred to ensiform cartilage and to the back.

Vomits soon after food. Bowels confined.

Somewhat anæmic.

I cannot yet say whether the tendency for these areas to appear on the right side in stomach affections has any significance.

I do not think it is possible at present to say whether the appearances of one or other of these areas of tenderness has any value in differential diagnosis. However the uppermost one (seventh dorsal) seems particularly to appear in anything which causes vomiting [compare Case 1, page 11], and when it appears as a sequel to vomiting it is frequently accompanied by the area next above it, corresponding to the sixth dorsal. Now we saw above that this sixth dorsal area was associated with disease in the lower part of the œsophagus, and hence I feel inclined to think that when both areas are present the disease is somewhere near the cardiac orifice. In the same way the ninth dorsal area is shared by both the stomach and intestine, and thus a lesion of the stomach causing pain over this area is probably situated nearer to the pyloric orifice; I possess, however, no records of *post-mortem* examinations to prove this, and at present it is a pure supposition.

In cases of gastralgia and gastic ulcer in which the pain bears a definite relation to the ingestion of food I think I am justified in making the following statement as regards the length of time after food at which it comes on. In those cases where the seventh dorsal segment is affected and the maximum tenderness is situated at and around the ensiform cartilage the pain comes on within half an hour after the food is swallowed. On the other hand in cases where the ninth dorsal segment only is affected the pain does not come on until the food has been swallowed at least an hour. This again tells in favour of the view that the seventh dorsal segment supplies the cardiac end of the stomach whilst the ninth dorsal supplies the pyloric portion.

Thus the areas which are affected in disturbances of the stomach correspond to the supply of the seventh, eighth and ninth dorsal segments.

Intestine.

The pain of intestinal affections is frequently quite diffuse and ill-defined. In fact it is not referred at all. Such pain is unaccompanied by tenderness in the skin. On the other hand a considerable number of cases of intestinal disturbance exhibit well marked pain and tenderness. I watched a large number of cases of diarrhœa which occurred in out-patient practice, and, as far as my experience goes at present, found that those cases in which "rolling," "griping," "doubling up pain" was complained of there was no pain in the back and no cutaneous tenderness. On the other hand a fair number of cases of acute diarrhœa, either due to some direct irritant, such as improper food, or more rarely to the disease known as "summer diarrhœa" were associated with pain below the umbilicus and over the twelfth rib, marking out to a greater or less extent the area supplied by the tenth dorsal segment. In those cases associated with a direct history of irritant food, I found the area corresponding to the ninth segment was also tender. If vomiting occurred in association with this diarrhœa, all the areas from the seventh to the tenth dorsal inclusive, might be present to a greater or less extent.

On the other hand the majority of cases of summer diarrhœa in which the stools were like "black water" complained of no referred pain, and did not show cutaneous tenderness.

It is also noticeable that cases of lead colic do not complain of referred pain and do not show cutaneous tenderness, even when the onset is fairly acute, and the "griping" pain well marked.

I feel inclined to think the pain and aching in the back loins and abdomen which appear in typhoid fever, is sometimes referred intestinal pain. In the case 52, there was at one time well-marked tenderness in the skin corresponding to the 10th dorsal area only. I have since seen several cases of typhoid fever, in which there was well-marked cutaneous tenderness over the abdomen and loins on both sides at the end of the second week of the disease.

Fig. 28 is taken from a man of thirty-nine, who suffered from dysenteric diarrhœa with fluid dark brown offensive stools, frequently containing blood. He was much relieved by enemata of creolin, but just after the injection he suffered a good deal of pain accompanied by exquisite cutaneous tenderness as shown in the figure. So tender was his abdomen, that he was obliged to have the clothes supported by a cradle. The tenderness varied from time to time; sometimes it occupied only the area of the tenth dorsal segment, sometimes that of both the tenth and eleventh segments. Pressure relieved this pain, and to show how purely superficial it was, I may mention that it was completely removed by an ointment containing thirty grains of cocaine to the ounce of lanoline.

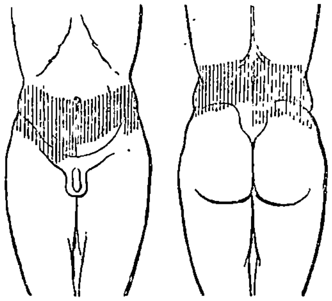

FIG. 28.

To show the distribution of cutaneous tenderness in Case No. 29. The area on the left side represents the 10th dorsal; that on the right side the 10th and 11th dorsal.

Case 29. Edward B., aged 39, compositor. (U. C. H., Dr. Roberts).

Eight months before admission had an attack of diarrhœa in which he passed blood. Lasted six weeks. Diarrhœa never quite ceased.

On admission pale, very thin, skin dry.

Appetite good, tongue large, flabby, not furred. No nausea or vomiting. Stools fluid, dark brown, very offensive. Contained altered blood in considerable quantities. Sometimes stools were quite fluid, sometimes they contained a few hard lumps. Abdomen distended.

No physical signs in lungs or heart.

Complained of a great deal of pain in the abdomen below the umbilicus, and in the back below the twelfth dorsal spine.

There is great cutaneous tenderness, worse at one time than at another. Always situated in the areas of the tenth and eleventh spinal segments, but sometimes worse on one side, sometimes on the other. So tender is he, that the bedclothes are supported by a cradle. Yet firm deep pressure relieves, rather than aggravates, his pain.

Under creolin enemata and oil injections he improved, and left the hospital, passing liquid stools free of blood and free from pain. Tenderness absent.

Died at home two months afterwards.

In this connection a case of malignant stricture of the splenic flexure (autopsy) is instructive. The maximum tenderness was over the tenth dorsal with some tenderness over the eleventh dorsal area. These areas were I take it, of intestinal origin. In addition she suffered from very marked dyspepsia with flatus and occasional vomiting, and both the seventh, eighth and ninth areas are more or less tender (stomach). In addition she developed bronchitis with troublesome cough and expectoration, and it was from this that she actually died. With the advent of bronchitis she developed great tenderness in the upper part of the back, between the spine of the scapula and the third to fourth vertebral spines, the point corresponding to the second dorsal area.

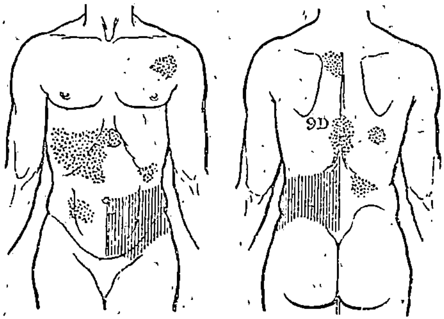

Fig. 29.

To show the distribution of cutaneous tenderness in a case of stricture of the sigmoid flexure of the large intestine (Case No. 30). The shaded area represents the 10th and 11th dorsal. The dotted areas are the same on the opposite side, and represent its maximum spots. The dotted area in the region of the liver represents the 7th and 8th dorsal. The dotted areas in the region of the spine of the scapula, and the front of the chest represent the maxima of the 2nd dorsal.

Case 30.—E. W., aged 46, U. C. H. (Mr. Heath), June, 1891.

Four years ago began to feel "fulness" in lower part of bowel.

Three years ago motions began to get smaller and passage gave her pain. Constipation alternating with diarrhœa.

November, 1890, passed blood from rectum. This has continued on and off ever since.

Losing flesh lately.

On admission abdomen much distended. Marked cutaneous tenderness over left half of abdomen below umbilicus as in fig.

28. This was probably due to the distension of the gut produced by the stricture and not to the destruction caused by the malignant disease itself. Some ill-defined tenderness on right side. Liver felt 3in. below costal border.

Considerable cutaneous tenderness in the region of the liver and back as in fig. 29.

Early pneumonic signs. Large râles and increased vocal resonance, especially left side. Much cough and some expectoration.

Note the spots of tenderness in the upper part of chest.

June 17. Difficulty in passing urine, and diarrhœa came on.

August 1. Died somewhat suddenly.

Post-mortem.—Intestines matted with recent lymph. Fluid in abdomen. A large mass of new growth involves sigmoid flexure. Large intestine and cæcum distended. Liver much enlarged; many small secondary growths. Gall bladder contains large number of stones. Left pleura full of fluid. Left lung collapsed.

Thus a certain number of cases in which the intestine from the pylorus to the end of the colon was affected showed definite cutaneous tenderness, corresponding more or less to the ninth, tenth, eleventh and possibly twelfth dorsal areas.

Liver and Gall Bladder.

The pain of liver disease is difficult to work out satisfactorily, for in those cases which are accompanied by pain the diagnosis of uncomplicated liver disease is not always satisfactory. Moreover a large number of cases of disease of the gall bladder which come to operation or autopsy are in reality secondary to malignant disease. In this connection the following case, for which I am indebted to Dr. Ringer, is very instructive. He was aged 47, and in July '91 began to suffer for the first time from severe attacks of biliary colic followed by jaundice. In January he was admitted, suffering from considerable pain, with cutaneous tenderness over the eighth and ninth dorsal areas on the right side, and the sixth and eighth dorsal areas on the left side. The tenderness was well defined, and on his left side the curious relation borne to one another by the sixth and eighth dorsal areas in the axillary line was beautifully shown. He was not jaundiced, but there was a trace of bile in the urine. A mass could be felt under the right costal margin, which was rightly supposed to be gall bladder. Mr. Pollard operated and removed 138 gall stones. All the old pain and tenderness disappeared and never returned, but secondary growths made their appearance in the lungs and skin, and he died on February 11th. A large mass of new growth was found in the portal fissure, blocking the bile ducts. Now although there was malignant disease I cannot help thinking that the pain and tenderness were due to the gall stones, for the pain exactly resembled that in cases of biliary colic in which gall stones pass, and it disappeared entirely after the operation although the malignant disease was untouched.

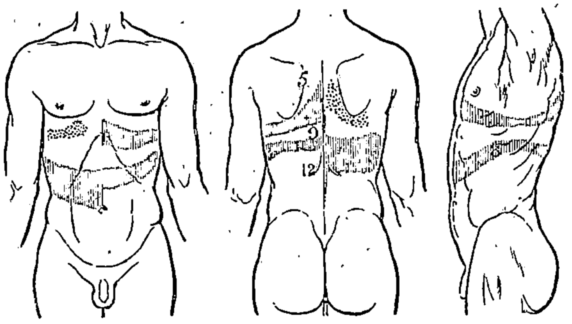

FIG. 30.

To show the extent of cutaneous tenderness in a case of hepatic colic (Case No. 31).

The areas represent the distribution of the 6th and 8th dorsal on the left side, and the 8th, 9th, and part of the 6th dorsal on the right side.

The figures on the posterior view are placed at the level of the spines of the vertebræ. The figure 8 on the lateral view is placed over the 8th intercostal space.

Case 31.—Frederick C., aged 47, tailor. U. C. H. (Dr. Ringer).

July, 1891. Several attacks of gallstone colic followed by jaundice. The attacks came on daily or there might be a week between the attacks. In the intervals pain in the right side. Stools greyish white during the attacks. Was perfectly well up to July.

Jan. 1892, admitted. Emaciated and pale. No jaundice. Continuous aching pain in right hypochondriac region. Tongue furred. Liver not felt. Below ninth costal cartilage an indistinct mass can be felt. Bowels confined. Motions normal colour. Urine contains a trace of bile. No vomiting.

Intense cutaneous tenderness so great that the head of a pin is felt as a sharp point, and he says that he is being pricked when touched over the areas marked in fig. 30.

Jan. 9. Mr. Pollard operated. Extensive adhesion round gall bladder.

Jan. 14. Mr. Pollard removed 138 gall stones from the bladder.

Jan. 15. All old pain gone. No sign of cutaneous tenderness. Slight pain in the wound only.

Feb. 9. Cough. Pneumonic signs left lung.

Feb. 11. Died.

Post-mortem.—Bile ducts surrounded by dense firm tissue. At portal fissure large mass of new growth. Growth limited very closely to Glisson's capsule. No scattered masses in liver. No new growth in gall bladder. Common bile duct and cystic duct were patent. Numerous small growth in omentum. Stomach, intestine, spleen, kidneys normal. Lower lobe of left lung solid, Pneumonic patches ; gangrene of extreme lower part.

The well known pain in the back at the level of the eleventh dorsal spine extending outwards to the line of the angle of the scapula and associated with pain "over the gall bladder" are simply the maximum spots of the eighth dorsal area. The eight dorsal segment seems to be particularly associated with the gall bladder and I believe it is for this reason that this area shows a greater readiness to appear on both sides in biliary colic than any of the other areas associated with the liver.

In this description of the pain and tenderness in association with gall stones I am in almost complete accord with Mackenzie.

In some cases of liver disease such as the enlarged and congested liver of primary, and not of secondary, origin the tenderness takes one segment lower than in cases of gall stones. The whole of the tenth dorsal area is more or less tender. Makenzie expresses a doubt as to whether this is not due to perihepatitis but I think I have satisfied myself that there is this cutaneous tenderness between the twelfth rib and the crest of the ilium. These cases also show the spot in the eighth space and mid-axillary line so common in liver disease.

Again, disturbances of the liver are associated with pain and sometimes tenderness over the shoulder joint and over the neck corresponding to the distribution of the lower roots of the cervical plexus; but I have not yet satisfied myself of the diagnostic significance of this pain in the lower part of the neck.

Thus in disturbances of the liver the pain is referred to, and the tenderness appears over, the areas supplied by the eighth and ninth dorsal segments and sometimes over that of the tenth dorsal on the right side. If the gall bladder is affected the pain and tenderness also lie over the eighth and ninth dorsal areas but the fifth, sixth and seventh are as a rule also implicated more or less.

Kidney and Ureter.

The pain of renal calculus is most useful for this investigation; for the diagnosis of stone in the kidney is practically always confirmed by operation.

The pain is said to be situated about the tip of the twelfth rib in the loin behind, and over a point in the abdomen internal to the crest of the ileum. During an attack of colic the pain is said to shoot round "the lower part of the back" (about the first lumbar spine) to the front of the abdomen and thence to the thighs and even down to the inner side of the knee. This pain is associated with marked cutaneous tenderness especially after an attack of colic. After an attack the patient complains that he feels "bruised, just as if he had been beaten about the back and loins." Mostly the area of tenderness is the tenth dorsal but sometimes the eleventh and twelfth dorsal areas are affected. Occasionally even some portion of the first lumbar with its spot on the inner side of the knee is marked out in tenderness.

One patient gave me the following interesting account showing the difference between an attack of renal colic in which no stone passed and one in which he actually passed a stone.

I first saw him just after an attack. The pain was located over the tip of the twelfth rib and on part of the crest of the ileum. The whole loin was sore and tender over an area marking out the tenth, eleventh and twelfth dorsal areas. No calculus had passed. Sometime before this he had had an attack which was similar excepting that the pain gradually spread from the region of the tenth dorsal to the upper sacral region and to a point in front just above Poupart's ligament 2 to 3 inches from the middle line (maximum of eleventh dorsal). Then it spread down the inner side of the thigh to the knee; then to "the bottom of the back" through the perineum to the urethra. In this attack his doctor said he passed a uric acid calculus.

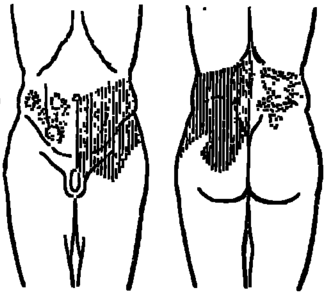

FIG. 31.

To show the distribution of cutaneous tenderness in a case of severe renal colic (Case No. 32).

The shaded area on the left side represents the 10th, 11th, and 12th dorsal. The dotted area on the right side represents the less defined tenderness associated with the presence of the maxima of the 10th and 11th dorsal areas.

It is interesting to note that this case showed a rather wider distribution downwards [11th and 12th dorsal] than is usual in cases of renal calculus in the inter-paroxysmal period. This was possibly due to the impaction of the stone in the ureter.

Case 32.—Thos. D., aged 37. U. C. H., June 30, 1891 (Mr. Hill).

Eight years ago passed a stone of a slate colour and the size of a bean. Pain came on after a long railway journey and lasted for twenty-four hours when the stone passed.

Was free from pain for a year and then began to pass crystalline masses, some of which were the size of a pea and were covered with minute points. On analysis they were said to be uric acid.

For three years he had about one attack of pain a year and passed small calculi. Lately the attacks have been more frequent. Blood always appears in urine after passage of calculus.

When I saw him he had just had an attack of colic, but no stone had passed. The pain starts behind at about the tip of the twelfth rib and runs forwards to a point in the left iliac fossa. Thence it passes to an intense centre of pain situated just above the internal ring. Thence it passes to another very painful focus in the pubic hair close to the root of the penis. Thence it runs down the inner side of the thigh to end at about the lower third of the inner aspect above the knee. As soon as the pain reaches the point over the pubes retraction of the testicle takes place. He says the pain is like that of cramp and at the same time there is pain as if he had taken a strong purgative.

During the paroxysm certain points are excessively sore. The testicle is very painful and tender, but he says the scrotum is never sore.

When I first saw him he was suffering from great pain in the left loin and lower part of abdomen and groin.

There was marked cutaneous tenderness with exaggerated superficial reflexes on the left side as in fig. 31. There was also some tenderness of a less defined character in the right loin and abdomen.

This cutaneous tenderness does not affect the scrotum except perhaps at its extreme upper part, *i.e.* the greater part of the skin of the scrotum is not tender. But the left testicle is intensely tender, and he cannot bear even the slightest touch.

July 1. Mr. Hill removed a calculus weighing six grains from the left ureter.

Seen again April, 1892. Wound healed. Has remained free from pain. No tenderness.

Case 33.—Geo. H., aged 24. U. C. H., June, 1891 (Mr. Hill).

Pain in left loin and across front of abdomen for five to six years. Agonizing attacks about once a month. Blood in urine, especially after an attack.

On admission.—Abdomen flaccid. Lower end of left kidney felt; does not seem to be enlarged.

Urine: amber coloured; acid. Microscopically contains blood and urate of soda.

The pain radiates from the tip of twelfth rib behind to a point in the left iliac fossa about midway between the anterior superior spine and the umbilicus and a little below the level of the umbilical line.

Definite cutaneous tenderness over the area of the tenth dorsal spinal segment. The tenderness is especially marked over the tip of the twelfth rib behind, and over the point on the left iliac fossa to which he refers his pain.

June 17. Mr. Hill removed an oxalate of lime calculus from the left kidney. It lays free in the pelvis and weighed twenty-seven grains. Dark coloured, ovoid stone, flattened from side to side and studded with small nodules.

Oct. 28. Has remained free from pain. No tenderness. Is in excellent health. Urine normal. No blood. Scar looks perfect.

In a patient with a floating kidney, who had no pain or tenderness as he lay in bed, I have seen the characteristic pain at the tip of the twelfth rib with cutaneous tenderness over the area of the tenth dorsal, produced by the rough manipulation of an examination.

Thus the kidney is particularly associated with the cutaneous areas of the tenth, and to a less extent those of the eleventh and twelfth dorsal and first lumbar areas. On the other hand, diseases of the pelvis of the kidney and of the ureter, seem to be particularly associated with the eleventh and twelfth dorsal and first lumbar areas.

Testis.

The testis is not so satisfactory as the kidney, owing to the great tendency of diseases of this organ to produce "spreading." (*Vide* chapter iv.)

In the case of a youth in whom one testicle had not descended, but lay in the inguinal canal, I made the following observations. In the course of his work he knocked it against a bench and it swelled and became painful. The pain was situated at the tip of the twelfth rib behind, and just internal to the crest in front, oddly enough on the opposite side, mainly, to the affected testicle. There was great superficial tenderness over the area of the tenth dorsal segment.

Case 34.—George D., aged 16. U. C. H., Jan., 1891 (Mr. Barker).

Is a carpenter, and pressure on the bench causes great pain in the undescended right testicle.

No testicle in right half of scrotum, but just to the inner side of the external iliac artery is a soft roundish swelling. When pressed upon it gives him testicular sensation. When he stands up index finger can be inserted into external abdominal ring, and the lower end of the swelling can be distinctly felt. In the erect posture it comes lower and can be pressed against the crest of the pubes. This gives him great pain.

The testicle itself is very tender, but the cutaneous tenderness on the occasion on which I saw him marked out the tenth dorsal area on the left side (not the right as might have been expected).

Mr. Barker removed the testicle, and he left the hospital Feb. 5, free from pain.

Again in cases of true traumatic orchitis when acute, there is intense pain over the loin and sometimes tenderness in the area of the tenth dorsal segment.

Thus the testicle seems to be peculiarly associated with the tenth dorsal spinal segment.

Epididymis.

Epididymitis is mostly gonorrhœal in origin and is associated with considerable pain when acute. In an ordinary moderately acute case the pain is situated over the fifth lumbar and first sacral spines behind and just over about the internal inguinal ring in front. These spots are generally tender and correspond to the maximum of the eleventh dorsal area. Sometimes the pain and tenderness are as in fig. 32, and thus correspond to both the eleventh and twelfth areas.

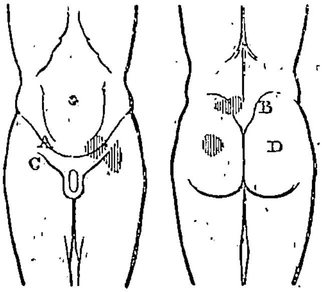

FIG. 32.

To show the cutaneous tenderness, and at the same time the seat of the pain and aching in a case of gonorrhœal orchitis (Case No. 35).

The spots marked A and B, correspond to the maxima of the 11th dorsal area. The spots marked C and D correspond to the maxima of the 12th dorsal area.

Case 35.—S. K., aged 28. U. C. H., casualty department, May, 1892. Had a "running" for nine weeks. It stopped a week ago, and next day swelling came on in "privates." Now well marked epididymitis. Left epididymis swollen to size of small apple, and very tender.

Pain passes from the point D (fig. 32) to B, and thence round the crest to the lower part of the abdomen and upper part of the groin. It is not sharp or shooting, but is dull and aching, "as if someone had kicked him." Cutaneous tenderness, as in fig. 32.

It is a noticeable and instructive fact that although the epididymis is exceedingly tender to manipulation, there is no cutaneous tenderness of the scrotum. Thus there is, frequently considerable cutaneous tenderness over the outer border of Scarpa's triangle in a situation far removed from any of the affected structures, but no cutaneous tenderness over the epididymis itself [*i.e.*, scrotum], excepting at the upper portion, where the skin is supplied from the twelfth dorsal segment.

If the epididymitis is very acute, the tenderness may become very extensive, reaching to the inner side of the knee and the outer side of the thigh, and it may even spread into the perineal area, jumping from the lumbar supply to that of the sacral areas.

Case 36.—Peter W., aged 40. U. C. H. (Mr. Heath), August 30, 1891. Denies gonorrhœa or syphilis.

August 27. Gnawing pain in left testicle.

August 30. Considerable swelling of left testicle, both body and epididymis. Epididymis hard, nodular, tender. Body also swollen, a little harder than natural and very tender. Spermatic cord as a whole thickened. Skin of scrotum reddened, and a little œdematous. No urethral discharge. Prostate normal in size and consistency. Temperature rose to 100.6.

Pain starts from the lower lumbar and upper sacral region, extending laterally on to the buttock. It also extends down the outer and inner aspects of the left thigh, and on to the abdomen above the pubes.

Intense cutaneous tenderness marking out the eleventh, twelfth and first lumbar areas.

September 7. All pain gone excepting in groin. No marked tenderness.

Thus the epididymis seems to be particularly associated with the 11th and 12th dorsal segments.

Bladder.

Erichsen states that the pain of stone in the bladder "may be experienced not only in the region of the bladder and perineum, but may radiate widely in the course of the nerves of the lumbar and sacral plexus, the patient complaining of a heavy dragging sensation in the groins, extending down the outside and back of his thighs and not uncommonly experienced in the soles of the feet."

I have met with no case, so far, which showed this wide distribution. The patient usually complains of pain in the perineum—pain at the end of the penis and pain over the ischial tuberosities. Sometimes this pain is accompanied by tenderness. Fig. 33 shows that this pain represents the gluteo-perineal (third sacral) area, for the patient complains exactly of this pain, and the tenderness occupied the area in the figure. The pain was caused by the unfortunate removal of a piece of mucous membrane of the bladder, during the crushing of a phosphatic calculus.

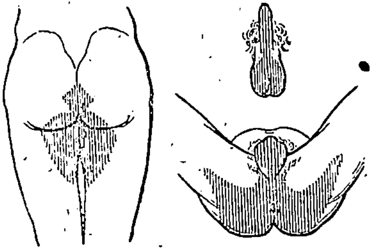

FIG. 33.

To show the cutaneous tenderness in a bladder case (Case No. 37).

The shaded areas represent the 3rd and 4th sacral. Note that there is a small patch of skin at the base of the scrotum which is unaffected. This is supplied from the 12th dorsal.

Compare with this figure the distribution of the eruption shown in fig. 10.

Case 37.—Wm. Thos. C., aged 51. U. C. H., May, 1892. Twenty years ago gonorrhœa. Two years afterwards developed a stricture. Up till six months ago had been in the habit of passing a catheter.

April 24. Could not pass any urine. No instrument could be passed into the bladder. Under opium and hot baths, No. 5 French passed the same evening. Urine alkaline, foul mucus, pus crystals of triple phosphates and micrococci.

He was slowly dilated, and on May 23 calculi were detected in the bladder.

May 24. Complains of pain before and during the passage of urine, but none afterwards. The pain is situated a little to the left of the anus and in the perineum. There is no cutaneous tenderness.

May 25. Two small stones ($\frac{3}{4}$ inch and $\frac{1}{2}$ inch in diameter) were crushed. The second stone gave considerable trouble, and on more than one occasion a small piece of mucous membrane was brought away in the blades of the lithotrite. Bleeding was considerable.

May 26. 9 a.m. Rigor.

May 27. Frequency of micturition; every hour and a-half to two hours. No blood; some pus and mucus.

May 28. Frequency of micturition continues. He complains of over the following points:—

[1st] Over the coccyx.

[2nd] Over a point on the inner aspect of both thighs, just external to the outer border of the perineum.

[3rd] Pain also in middle of perineum, "running down the passage" to the tip of the penis.

There is no pain above the pubes.

There is intense cutaneous tenderness in the extreme lower part of the spine, and in the perineum, as in fig. 33. The skin of the penis and scrotum is tender. Temperature normal.

May 29. The cutaneous tenderness was marked out again, and coincided with that in fig. 33.

June 3. Temperature normal since May 27. Another small stone (size of hazel nut) was crushed. Several small portions of soft tissue were removed from the bladder by the evacuator, evidently pieces of mucous membrane which had remained in the bladder since last operation.

Tenderness over a similar area was also produced by the accidental use of carbolic lotion instead of boracic, to wash out a patient's bladder. The whole glans, which had not been touched by the fluid, was intensely sore. Thus the ordinary pain due to lesions of the mucous membrane, seemed to correspond to the areas of the fourth and third sacral segments.

In cases of retention, however, an additional pain is found. Suppose a patient suffers from simple obstructive retention, the result of a stricture in a man, or better still, the tilting forwards of a fibroid or of the pregnant uterus in a woman, the bladder gradually fills, and a dull aching pain appears over the lower part of the sacrum, passing into the perineum. This pain is more or less continuous, and is described by women who have borne children as like the pain "at the very bottom of the back" in labour. Every now and then the patient is overcome by an irresistible desire to pass water and strains against the obstruction. And now a quite different pain appears, of a sharp, shooting character, radiating from the lower lumbar and sacral regions behind, round the ileum to the lower part of the abdomen just above the pubes, and also down the inner side of the thigh to the knee. This lumbar pain soon goes, only to return again after an interval; but the sacral pain seems to be continuous. In all my cases the pain was completely relieved when the urine was drawn off. One of my female patients had retention several times, owing to the displacement of a large loose fibroid, causing pressure on the neck of the bladder. Each time she gave exactly the same description of the dual nature of her pain. She was always relieved by the passage of a catheter. The amount drawn off varied on different occasions from 40 to 60 fluid ounces.

Case 38.—Mary D., aged 34 years, married. U. C. H., obstetrical department.

August 22. Complete retention of urine for nearly twenty-four hours. Bladder reached to within an inch of the umbilicus.

She was in intense pain. The pain radiated from the lower part of the sacrum and coccyx into the perineum. It resembled a labour pain, and was accompanied by a feeling of weight and "bearing down" in the perineum.

When she strained to pass water this pain was joined by a pain over an area extending from the region of the fifth lumbar and first sacral vertebræ outwards, between the great trochanter and the crest of the ileum to the lower part of the abdomen and groin. It also extended on to the outer side of the thigh, and also down the inner side of the thigh to a point a little below the junction of the middle and lower thirds of the femur. This pain rapidly passed off, but recurred again and again whenever she tried to pass water.

No cutaneous tenderness.

Forty-one ounces of urine drawn off. Pain immediately relieved.

A large fibroid was felt, which had fallen forwards over the neck of the bladder.

She came up on two subsequent occasions, with retention from the same cause, and always complained of the two forms of pain.

Thus lesions of the mucous membrane of the bladder affect the lower sacral areas (third and fourth), whilst the pain of ineffectual contraction is difused over the eleventh and twelfth dorsal and the first lumbar areas.

Prostate.

Before I leave the male urino-genital tract I must mention the pain caused by diseases of the prostate. I have reserved this section to the last, owing to the wide distribution of the pain in diseases of this organ.

The most marked pain is, as usual, caused by acute inflammation of the prostate—acute prostatitis.

The highest pain is situated over the twelfth rib behind and just internal to the crest in front. Pain may be present over the fifth lumbar spine behind, and also over the internal ring area in front, but I have no case where any lower dorso-lumbar areas were affected.

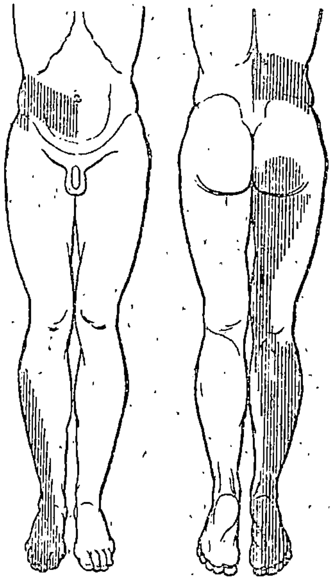

FIG. 34.

To show the distribution of cutaneous tenderness in a case of acute inflammation of the right lobe of the prostate (Case No. 39).

The uppermost area represents the supply of the 10th dorsal segment. The area on the back of the leg represents the 3rd, 2nd and 1st sacral from above downwards, and the area on the outer side of the leg the 5th lumbar.

But the patient also has pain over the lower part of the sacrum, and the ischial tuberosities over the upper part of the calves and over the soles of the feet, especially at the root of the great toe. One of my patients whose prostatitis was set up by improper instrumentation at the hands of a quack, said, " My life is a burden for I cannot stand owing to the pain in the soles of my feet, I cannot walk owing to the pain in the calves, sit on account of the pain over the ischial tuberosities and in the perineum, or even lie owing to the pain in my loins and side."

If the prostatitis is fairly acute tenderness is present over all the areas corresponding to this distribution of pain—the tenth and eleventh dorsal, the first, second and third sacral.

In the case from which fig. 34 was taken, the tenderness was very well marked and the surgical report was "right side of prostate only, enlarged and tender."

Case 39.—Thos. T., aged 52, shampooer, casualty U.C.H., August 23, 1891.

Five weeks before he had retention of urine from a large prostate. No. 10 catheter passed. Came up August 21st, second attack of retention. Catheter again passed. Next day great pain. August 23, pain referred to spots in fig. 34, with cutaneous tenderness. Right lobe of prostate swollen, enlarged, and very tender. No prostatic discharge. Six weeks afterwards under treatment had lost all pain and tenderness in the skin and right side of prostate had subsided.

Thus the pain and tenderness when present in prostatic disease seem to belong partly to the dorsal and partly to the sacral areas. The dorsal areas most often affected are those corresponding to the tenth and eleventh dorsal segments. The lumbo-sacral areas which are affected correspond to the first, second and third sacral with sometimes the fifth lumbar in addition.

Ovary.

Ovaritis is the bugbear of the gynecologist owing to the general disturbances it is liable to produce. I shall therefore not describe the pains of ovaritis in detail, but only such pain as can be distinctly traced to the ovary.

During vaginal examination no pain is caused by taking a normal ovary between the fingers but a curious sensation is produced apparently akin to "testicular sensation." If however the ovary is inflamed very definite pain is produced which the patient localises in the loin behind and pain is also frequently referred to a point just below and to one side of the umbilicus. This pain is frequently accompanied by tenderness of the skin over the area of the tenth segment. When obstetric assistant I frequently found cases of ovarit is quite without tenderness. But after examination these same cases often showed marked cutaneous tenderness corresponding to the tenth dorsal area and to that area only.

Case 40.—Annie D., married. U.C.H. (Dr. Spencer) Aug. 1891.

Well developed healthy woman.

Nothing felt from abdomen. Body of uterus retroverted. Both ovaries prolapsed and easily felt. Both tender, right one more so than left.

Pressure on right ovary causes pain in the loin a little to the right of the middle line at the level of the second lumbar vertebra and pain in front about 1½ inch to the right and 1 inch below the umbilicus.

Cutaneous tenderness especially after examination accurately marking out the supply of the tenth dorsal segment. It shows the little downward tag just external to the anterior superior spine on both sides. The lower border of the tender area is 2 inches above the pubes in the middle line.

Case 41.—Mrs. V. U.C.H. (Dr. Spencer), Aug. 21, 1891.

Constant pain in left loin and in abdomen below the umbilicus. At menstrual periods has pain in addition over the lower part of the abdomen and down the back of the leg.

A prolapsed ovary is felt lying directly behind the uterus. It is excessively tender. Even the lightest pressure causing pain. This pain is referred to a point in the left loin corresponding roughly with the tip of the twelfth rib. It is also referred to a spot in front a little to the left of and below the umbilicus.

Intense cutaneous tenderness marking out in a beautiful manner the tenth dorsal area.

Thus the ovary like the testis causes referred pain and cutaneous tenderness along the tenth dorsal area.

The Appendages.

It is needless to mention that pressure on normal tubes causes no pain. But in some cases of salpingitis very definite pain is caused by manipulation. This pain is situated over about the fifth lumbar and first sacral spines and "in the groin" above Poupart's ligament.

After removal of the ovaries the stump is sometimes tender. In the case I have figured fig. 35, some ovary and the whole tube was left on the one side (right) and the whole ovary and part of the tube removed on the other side.

Spontaneous pain was almost completely absent and the patient was bright and intelligent. After examination she had some pain and developed the tenderness over the areas in the figure (fig. 35). Thus pain and tenderness from the ovary seem to follow a line corresponding to one segmental area higher than that from the tubes. This I believe to be a valuable point in diagnosis.

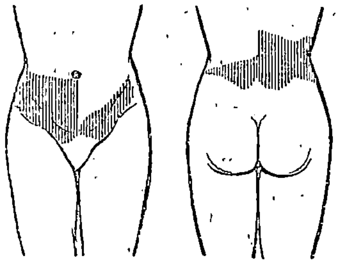

Fig. 35.

To show the cutaneous tenderness in Case No. 42.

The shaded area on the left side represents the 11th dorsal; that on the right side the 10th and 11th dorsal.

Case 42.—Eliz. B., widow. U.C.H. (Dr. Spencer), July, 1891.

In January, 1891, an attempt was made to remove both ovaries and both tubes for tubo-ovarian hæmatocele. The left ovary and greater part of the left tube were removed whereas a portion of the right ovary was left behind.

The uterus is movable, retroverted, and tilted a little to the right side. Right stump can be felt as big as tip of forefinger. On making pressure high up on the right side a tender body can be touched. Cervix rather small.

Strong, healthy looking woman, well nourished, bright, cheerful and not emotional. She complains of occasional pain, worse on right side than left in lower part of abdomen and back. Cutaneous tenderness well marked as in fig. 2.

Subsequently the whole of this pain and tenderness disappeared and she is now bright and well.

It is well known that after the removal of an ovarian tumour the patient complains of pain in the back over the region of the loin for which morphia is frequently given. It is customary to place a small pillow under the loin to ease this pain. In a case in which a tubal fœtation was removed by operation the tube and broad ligament were much injured and the nurse called my attention to the fact that the pain was not in the usual place but lower down than usual over the upper part of the sacrum. I made the same observation in a case where a cyst had been removed from the broad ligament. Both these cases had borne children and described the pain as like a "dull labour pain." I subsequently asked several patients from whom true ovarian tumours had been removed whether their pain resembled labour pain and got a distinctly negative answer.

Thus the Fallopian tubes, like the epididymis seem to be particularly associated with the 11th and 12th dorsal segments.

Cervix and Os Uteri.

It is a well recognised fact that erosions and early malignant disease of the vaginal surface of the cervix produce no pain. But directly they set up cervical catarrh or begin to invade the broad ligaments pain begins. Such a disease was therefore too wide reaching in its effects for my purpose.

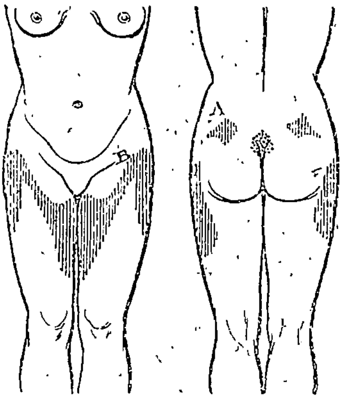

FIG. 36

To show the cutaneous tenderness produced by the passage of a probe in a case of cervical catarrh (Case No. 43).

The shaded areas marked A and B represent the 1st lumbar. The small dotted area over the sacrum represents the maximum of the 4th sacral.

I made several interesting observations during the copper sulphate treatment, in cases of cervical catarrh. The effects produced by the passage of the Playfair's probe were very instructive. As the probe enters the external os the patient complains of no discomfort. As it ascends the canal she begins to complain of a pain over the lower sacral region. As the probe advances still further the pain alters in position and is situated over the last lumbar and first sacral spines and in the groin in front. One of my patients repeatedly said:—"I first have pain at the very bottom of my back shooting through my legs and then as you push it further in I get a pain in my groin." The treatment was not in any sense of the word truly painful but it was frequently followed by some cutaneous tenderness in cases of cervical catarrh. (*Vide* fig. 36.)

Case 43.—Ellen T., aged 21, one child. U. C. H. (Dr. Williams), October, 1891.

Bright, well nourished, healthy looking woman.

Uterus, normal size. Slightly retroverted and retroflexed, but freely moveable. Right ovary can just be touched; not tender. Erosion on cervix. A good deal of clear mucus from cervical canal.

The erosion and cervical catarrh rapidly improved under the copper-sulphate treatment, and rest in bed.

October 22. Dressing the cervix was followed by cutaneous tenderness over the areas in fig. 36. She is now nearly well.

November 26. Free from pain and tenderness. Erosion healed. No discharge from cervix. Has passed through a menstrual period without pain. Has been at work for a week, and is bright and well.

In those cases where the canal is widely lacerated by previous labours, the lower (sacral) pain seems to be absent. In a case where a mucous polypus was very high up, not extending through the external os, the pain, accompanied by marked cutaneous tenderness, was situated over the area on the skin corresponding to the eleventh dorsal area together with the maxima of the twelfth dorsal.

This opens up the whole interesting question of dysmenorrhœa and its treatment, which I shall touch upon shortly later in this paper. (*Vide* p. 106.)

Thus the lower portion of the cervical canal seems to be associated with the 3rd and 4th sacral areas, whilst the upper portion in the region of the internal os is related to the areas of the 11th and 12th dorsal segments.

Pains during and after Labour.

During the first two stages of labour, pain appears over two separate points. The so-called "niggling pains" cause pain over the upper part of the sacrum and in the groins, whilst a pain appears later over the extreme lower part of the back in the region of the sacrum and coccyx.

The following case in which I induced labour at seven and a half months shows this very well, and gives us the clue to the meaning of these pains. The thin bougie was introduced in the evening, and I left her free from pain. Next morning, pain had come on over the last lumbar and upper sacral spines, shooting round the crest of the ilium on both sides to the lower part of the abdomen. Towards evening these pains were joined by pain over the lower part of the sacrum and coccyx, "shooting through the privates" to the mons veneris in front. I made a note at the time, "os dilating well," and I found that manual dilatation of the os caused increase of pain over the lower sacral area. By

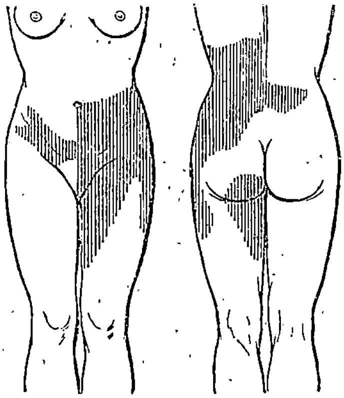

FIG. 37.

On the right half of the body on each of these figures is shown the cutaneous tenderness which appeared during the second stage of labour in Case No. 44. The area marked out represents the 11th dorsal.

On the left half of the body in each figure is shown the cutaneous tenderness which accompanied the "after pains" 48 hours after delivery in the same case. The areas represent the 10th, 11th, 12th dorsal and 1st lumbar, and the 3rd sacral at the back of the thigh.

the night, these lower pains had got the upper hand, and practically all the pain was situated over the lower part of the sacrum and coccyx and in the perineum.

"After pains" are caused by the uterus expelling clots, &c., that have been left behind at the time of labour; for they seldom occur if the uterus is well and carefully kneaded after the expulsion of the placenta. The pains are increased at the time by the administration of ergot. They are rythmical, coming on and passing off again, and each is mostly accompanied by the passage of a clot. They are frequently accompanied by considerable tenderness which is cutaneous and follows the areas laid down in this paper.

Case 44.—Mrs. L. U. C. H., Midwifery Department.

Second stage of labour. Membranes ruptured. Os dilated. Child presenting by vertex.

Pains round the body "like a hoop." Only in the groin and back. Very marked cutaneous tenderness as in fig. 37 (right half of each diagram). After delivery the pain and tenderness began to extend down the outer side of the thigh, and almost to the knee on the inner side of thigh.

Confined 48 hours. Complains of bad after pains. Marked cutaneous tenderness as in fig. 37 (left half of each diagram).

Third day after delivery all pain gone. Slight cutaneous tenderness still remains.

Recovered as usual, and was up on the tenth day.

Fig. 37 shows the cutaneous tenderness associated with the pain of the second stage of labour on the right side, and the tenderness found forty-eight hours after labour in association with "after pains" in the same woman. It will be seen that the pain of labour is associated with tenderness over the eleventh dorsal, and the "after pains" with tenderness over the tenth, eleventh and twelfth dorsal, first lumbar, and third sacral areas.

In multiparæ, the os is frequently extensively lacerated by previous labours so that the cervical canal practically does not exist. In such cases the lower or sacral areas of pain do not seem to appear after labour. In the case of a woman whose os was deeply lacerated by the birth of a large child the year before, "after pains" associated with cutaneous tenderness only appeared over the areas corresponding to the tenth, eleventh, and twelfth dorsal areas. There was no sign of reference to the sacral areas on the back of the thigh. She said that at all her labours before she was obliged to have someone to "rub her back" (over the lower part of the sacrum), but this time "she did not know herself," for she had no pain over any part of the back excepting where she "now has them."

It might perhaps be objected, that the few instances I have given of tenderness in the skin associated with after pains were open to doubt in that women were notoriously "hysterical." I think this objection is set aside by the fact, that out of 178 women whom I visited within 48 hours of labour, 52 had such well marked cutaneous tenderness, that I could map it out on a chart. Thus, in my experience of hospital patients delivered by students, 30 per cent. showed some cutaneous tenderness within the first 48 hours after labour. Moreover, my impression is that this tenderness is always worst in those cases in which the child was born before the arrival of the doctor, and in those cases in which the student was inexperienced (at the beginning of the month of duty), showing the relation of the pain and tenderness to incomplete evacuation of the uterus.

Thus the uterus like the bladder causes referred pain and cutaneous tenderness over a double set of areas. Anything which stretches the lower segment and especially the cervical canal proper causes pain and tenderness over the fourth, third and sometimes the second, and even first sacral areas.

On the other hand painful contractions of the uterus are accompanied by pain over the eleventh, twelfth and sometimes the tenth dorsal areas, together with occasionally the area of the first lumbar segment.

Pleura and Peritoneum.

Referred pain such as we have considered in this chapter is characterised by the following features.

1st. It tends to follow the lines of spinal segmentation rather than those of peripheral nerves. Thus on the chest it cuts across several ribs and does not follow the lines of any one intercostal space.

2nd. It tends to form bands round the body and thus in diseases of the abdominal viscera the pain may be quite as marked in the back as over the abdomen.

3rd. It is mostly associated with cutaneous tenderness, which may be quite as marked at a distance from the affected organ as in its neighbourhood. Thus gall stones are not only associated with tenderness over the area of the gall bladder in front, but also with tenderness over the ribs and spine behind. In the same way intestinal disorders may cause a band of tenderness extending round the body, not only in the loin but also overlapping the summit of the iliac crest on its way to the front.

Now I was long ago struck by the fact that in affections of the peritoneum the pain and tenderness differed fundamentally from that of referred pain, in that, although the pain is well localised, it does not follow the line of the nerve areas. Thus, for instance the patient will refer the pain to an area both above and below the umbilicus without at the same time complaining of any pain in the back or loin.

Moreover, there is no true cutaneous tenderness in peritonitis, and the tenderness that is present differs fundamentally in character from that which accompanies referred pain. If the skin only is picked up between the finger and thumb in a case of acute peritonitis the patient complains of no pain, but directly the deeper structures of the abdominal wall are included in the gentle pressure he complains of intense pain. Thus firm steady pressure as a rule relieves referred abdominal tenderness, whilst light pressure between the finger and thumb, or even the pressure of the clothes, at once elicits it. On the other hand in peritonitis light cutaneous stimulation causes no pain, but the slighest attempt at deep pressure, or even the lightest percussion, at once causes marked pain.

Again, the distribution of the two forms of tenderness differ fundamentally. The tenderness of peritonitis may be present all over the abdomen from the ensiform cartilage to the pubes and from one costal margin to the other, but it never appears at the angle of the scapula or over the sacrum and iliac crest. On the other hand, if referred tenderness extended over the area just mentioned, spots of tenderness would appear to a certainty over the ribs in the line of the angle of the scapula, over the costal margin in the eighth and ninth spaces, over the twelfth dorsal spine, over the twelfth rib, and over the last lumbar and first sacral spines. Thus the tenderness of peritonitis only makes its appearance over those points where pressure, &c., can actually affect the peritoneum, whilst referred abdominal tenderness is always present over bony points, where pressure cannot possibly produce any effect on the abdominal cavity or its contents.

Thirdly, we find that however great may be the pain caused by the peritonitis, the abdominal reflexes are not exaggerated as would have been the case if the tenderness had been cutaneous.

Case 45.—Amelia T., aged 42, married. U. C. H. (Mr. Beck). Was admitted November 27, for obstruction of the bowels.

Has never had any obstruction before, but for fourteen years has suffered from chronic constipation. Bowels open twice a week with medicine only.

November 20. Bowels last opened.

November 21. Sudden pain in lower part of abdomen, chiefly on the left side.

November 22. Went to work feeling ill.

November 24. Took medicine after which she vomited.

November 27. Admitted. Has not passed flatus or had desire to pass a motion since November 20. Had not vomited except after the purgative. Abdomen soft, but distended rather more on left than right side. Whole abdomen tympanitic to percussion.

Now it was a very striking feature, that although there was very marked tenderness of the left iliac fossa on even the lightest percussion or palpation all cutaneous tenderness was completely absent. There was no pain or tenderness in the back or loin, and the most posterior limit of the tenderness was a line from the tip of the eleventh rib to the crest of the ileum. Thus all tenderness was deep, and it was only present over those points where the peritoneal cavity could be disturbed by the manipulation.

It was thought to be a case of volvulus of the sigmoid flexure of the colon, and Mr. Beck operated at once. The colon and gut looked healthy, but patches of lymph could be seen on the coils of small intestine, and on separating these a little, thin pus escaped. On portions of the small intestine were well defined patches of lymph, and it was evident that these coils had formed the lining wall of an abscess cavity. This abscess reached down into the pelvis and was found to be connected with the left Fallopian tube. Vermiform appendix was normal.

Abscess cavity was drained and the woman did well.

I now give a case of peritonitis in connection with perforation of the vermiform appendix to illustrate the same point.

Case 46.—Mary Ann B., aged 49, married. U.C.H. (Mr. Barker), Dec. 5.

November 26. Pain in abdomen began, but she did not stop work. December 3. Became so severe she went to bed. Pills caused action of bowels.

She has long suffered from chronic constipation.

December 5. Admitted. Well nourished. Tongue furred. No vomiting. Pain after taking solid food. Abdomen not much distended. Nothing definite in either iliac fossa. No difficulty or pain on micturition. Nothing definite felt per vaginam.

Great tenderness and pain in the abdomen. The pain is situated in the right iliac fossa, and the tenderness extends from the upper part of the fossa to the costal margin above. It does not extend over the middle line or further back than a line joining the tip of the eleventh rib and the crest of the ileum. There is no pain or tenderness in the loin. The most marked feature is the complete absence of cutaneous tenderness. Thus the skin can be picked up between the fingers without giving her any pain, but palpation or light percussion cause considerable pain. Abdominal reflex not obtained.

December 10. She began to vomit. Mr. Barker operated and found a small abscess in connection with the vermiform appendix.

The appendix was perforated, but was not swollen, and except for the perforation was healthy.

December 16. Died. *Post-mortem.*—No general peritonitis Abscess cavity shut in by firm adhesions, lower end of abscess just below brim of pelvis.

In contradistinction to these two cases I give two cases of strangulated hernia in which cutaneous tenderness over the tenth dorsal area was very marked. Both cases showed well the tenderness in the loin which is present when the whole bandlike area of the tenth dorsal is present. The second case developed peritonitis after enterectomy, and yet tenderness did not re-appear after the operation.

Case 47.—Hortense D., aged 68, married. U.C.H. (Mr. Beck).

For six months has noticed a small lump in right groin.

January 6. In the morning noticed that the lump was larger than usual, and was painful. Enema, which she thinks brought away fœcal matter.

January 7. Vomited six or eight times. Admitted at noon.

Abdomen distended. Walls thin. In right groin slightly oval swelling, size of pigeon's egg. Obviously a femoral hernia.

There is no deep tenderness, but definite cutaneous tenderness marking out fairly the area of the tenth dorsal segment on the right side. Abdominal reflex on right side a little brisker than on the left side.

The usual operation was performed and patient did well.

Case 48.—Elizabeth W., aged 30. U.C.H. (Mr. Barker).

Six months ago first noticed a swelling in the right groin, which disappeared with rest.

Six weeks ago rupture came down again, but went back under rest.

December 22. Rupture came down again.

December 23. Began to vomit. Bowels opened naturally this morning for the last time.

December 24. Worse. Vomiting continued.

December 27. Vomited last at 6 a.m. Admitted to U. C. H. General condition on admission very satisfactory. Obvious femoral hernia in right groin. Abdomen not distended. No tenderness on percussion or palpation. Very definite cutaneous tenderness on taking up the skin of the abdomen between the fingers. This cutaneous tenderness marked out very beautifully the tenth dorsal area on the right side, and was so definite, that it could be demonstrated several times in succession to onlookers. There was no cutaneous tenderness over the hernia itself. Thus all the tenderness was at a distance from the affected part. The superficial reflexes over the tender area were distinctly exaggerated and the head of a pin felt like a point.

She refers her pain to a point just to the right, and a little below the umbilicus (*i.e.*, the maximum of the tenth dorsal area in front). She also has some pain in the loin and back.

Mr. Barker operated, and found that the knuckle of gut in the hernia had ruptured in two places, allowing a small quantity of fœcal matter to escape. He excised a portion of gut, and sutured the two ends.

December 31. She passed flatus freely, and her bowels were opened. Seemed to be doing well.

January 4. Complained of pain in the wound and right hypochondrium, but there was no cutaneous tenderness.

Temperature began to run up for the first time, and she died in the night.

Post-mortem.—The juncture of the two ends of gut had leaked and there was general peritonitis.

Now an exactly similar law holds good for the pleura. Every physician is familiar with the numerous cases of "pain in the side" increased by breathing and coughing which are unaccompanied by the physical signs of pleurisy. Such pain is mostly associated with cutaneous tenderness and is in reality referred. In order to render clear the difference between the pain of pleurisy and this referred pain let us consider the following example.

A man of 36 complained of cough accompanied by intense pain over an area the size of the palm of the hand situated over the sixth, seventh and eighth ribs. It extended from the level of the posterior axillary fold forwards nearly to the costal margin on the left side. There was no tenderness when the skin was lightly picked up between the finger and thumb but the most exquisite tenderness on the lighter percussion or pressure with the fingers. There was no tenderness in the abdomen or in the line of the angle of the scapula and over the vertebral spines. Over this painful area very marked pleural friction was audible. Now if this pain and tenderness had been referred it would have corresponded to the maximum of the eighth dorsal area. But with such exquisite pain the posterior and also the abdominal portion of this area must have been affected, and true cutaneous tenderness would have certainly manifested itself. I have more than once had the opportunity of seeing true referred pain accompanied by cutaneous tenderness on the one side associated with the local pains and deep tenderness of pleurisy on the other.

Another feature of the pain of pleurisy which I wish to emphasize is its conduction along the line of the intercostal nerves as opposed to those of the segmental areas. Thus a pleuritic pain situated in the ninth and tenth space in the line of the angle of the scapula will radiate forwards along these inter-spaces to about the umbilicus. From before backwards it will cut across the lines of the seventh, eighth and ninth segmental areas and will finally enter the distribution of the tenth dorsal segment. Although it radiates on to the abdomen all tenderness ceases as soon as the costal border is passed.

Case, 49.—James P., aged 56. U. C. H. (Dr. Roberts), 1892. Was at work and appeared healthy until two weeks before admission, when he developed a bad cough. The day before admission he coughed up blood, and when admitted, was still bringing up very highly blood-stained sputum.

Constant hard cough. Cough caused pain over an area the size of the palm of the hand, situated over the sixth, seventh, and eighth ribs. It extended from the level of the posterior axillary fold forwards nearly to the costal margin. There was no superficial or cutaneous tenderness, but even light percussion or pressure with the fingers caused pain. No tenderness in the abdomen in front of the costal margin or at the angle of the scapula, and over the vertebral spines.

Left base moves less than right. Over left base in the axillary line from the fifth rib downwards, very marked pleural friction. Slight signs of consolidation at left apex and base.

This pain was removed by strapping the left side.

Thus in conclusion I think we can formulate the following statement. Affections of the serous cavities of the body do not cause referred pain or cutaneous tenderness, but produce local pain which follows the lines of peripheral nerves and is associated with deep tenderness over the affected point only.

CHAPTER IV.

THE SENSORY FIBRES IN THE SYMPATHETIC SYSTEM.

I have gone through all the organs systematically in the last chapter and attempted to show that the pain and tenderness caused by certain disturbances in their normal activity is referred along the distribution of certain somatic nerves. Now on Ross's[1] hypothesis a knowledge of the nerve roots along which reference takes place enables us to map out the sensory supply which the affected organs receive by means of the sympathetic. For on this hypothesis the viscera receive their sensory fibres from that segment of the spinal cord from which the somatic sensory roots arise along which the pain is referred.

I will therefore put together the results arrived at in the previous chapter in the form of a table. But I must first call attention again to the fact that certain organs have a tendency to refer into the supply of the cervical plexus and that of the cranial nerves. The organs in which this feature is more marked are the lungs, heart, liver, intestines, and stomach. I cannot now enter into a full discussion of the significance of this fact, but must reserve it for my next

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[1] BRAIN, January, 1888.

communication in which I hope to describe the areas of referred pain and tenderness for the head and neck. I must however, for the purpose of this table state that I consider this reference to the head and neck to represent the supply of the vagus and depressor, and thus in all those cases where the disturbance of an organ produces direct reference above the clavicles I have inserted these nerves in my tabular statement of the segments from which they are supplied.

Heart.—1st, 2nd, 3rd, dorsal segments.
Cervical plexus [= depressor ?]

Lungs.—1st, 2nd, 3rd, 4th, 5th dorsal.
Cervical plexus [= vagus ?]

Stomach.—6th, 7th, 8th, 9th dorsal.
Cardiac end from 6th and 7th.
Pyloric end from 9th.

Intestines.—[A.] Down to upper part of rectum.
9th, 10th, 11th and 12th dorsal.

[B.] Rectum.
2nd, 3rd and 4th sacral.

Liver and *Gall Bladder.*—7th, 8th, 9th, 10th dorsal.
Perhaps 6th dorsal.
Cervical plexus [= vagus ?]

Kidney and *Ureter.*—10th, 11th and 12th. The nearer the lesion lies to the kidney the more is the pain and tenderness associated with the 10th dorsal. The lower the lesion in the ureter the more does the 1st lumbar tend to appear.

Bladder.—[A.] Mucous membrane and neck of bladder.
[1st.] 2nd, 3rd, 4th sacral.

[B.] Over-distension and ineffectual contraction.
11th and 12th dorsal and 1st lumbar.

Prostate.—10th, 11th, [12th] dorsal.
1st, 2nd, 3rd sacral and 5th lumbar.

Epididymis.—11th and 12th dorsal and 1st lumbar.

Testis.—10th dorsal.

Ovary.—10th dorsal.

Appendages, &c.—11th and 12th dorsal and 1st lumbar.

Uterus.—[A.] In contraction,
10th, 11th, 12th dorsal and 1st lumbar.
[B.] Os uteri,
[1st], 2nd, 3rd, 4th sacral [and 5th lumbar very rarely].

It is obvious from this table that the sensory supply of the viscera bears a very curious resemblance to Gaskell's[1] scheme of their motor and inhibitory supply. I was therefore exceedingly interested to hear from Dr. Gaskell that my supply closely represented the results obtained by Edgeworth by anatomical methods. Edgeworth's paper in the May number of the *Journal of Physiology* showed how closely we were agreed and yet how different were our results on certain points. Firstly as to the differences, which I do not think are essential. Edgeworth dissected and examined microscopically the sympathetic branches in the dog. Now this animal possesses thirteen dorsal and seven lumbar roots. Edgeworth finds that the large medullated fibres which he believes to be the sensory fibres of the viscera come off from the first dorsal to the third lumbar roots inclusive. There is then a blank of three roots from which no such fibres come off followed by another outburst of these fibres from the region of the seventh lumbar and first and second sacral. Now in man there are but twelve dorsal and five lumbar vertebræ and I find that my method gives a supply of visceral sensory fibres from the first dorsal to the first lumbar segments with perhaps also very rarely the second lumbar. Then follows a blank of three roots followed by a lumbo-sacral outburst from the fifth lumbar and first, second, third and fourth sacral. Of these the fifth lumbar is only occasionally seen. Now I think that considering the different anatomical construction of man and the dog these differences are but of slight importance. And now to pass on to the points upon which we arrived at similar results. Edgeworth finds that the heart, lungs, stomach, liver, and intestines are all supplied by large fibres from above which reach them in the vagus, and in the case of the heart by the depressor nerve. Now these are the organs which I believe give reference into the areas above the clavicle, *i.e.*, into the region of the head and neck above the upper gap. The most definite reference into the neck is given by the liver, lungs, and heart.

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[1] *Journal of Physiology*, vol. vii., 1885.

Another point on which we are united as will be seen by comparing my table with Edgeworth's chart is in the double supply of the bladder. The double supply of the prostate and uterus he does not seem to mention. Thus I think that the results produced by both the anatomical (Edgeworth) and by the physiological methods tend to show that the sensory distribution of the sympathetic essentially follows the lines laid down by Gaskell for the course of its motor and inhibitory fibres.

Before I leave this subject I should like to mention some interesting points brought out by the recognition of the double supply of the bladder. In those cases where the distribution of the analgesia points to a lesion of the sacral region of the spinal cord the patient says he knows when he wants to pass water but he has no power of retaining it. As soon as a few ounces have collected in his bladder he "feels he wants to pass it and cannot hold it back." It then comes from him involuntarily. A patient in this condition has analgesia over the areas corresponding to the third and fourth sacral segments.

If, on the other hand, the analgesia is over an area corresponding to the eleventh and twelfth dorsal and first lumbar areas, and sensation is normal over the sacral area, the condition of the bladder is exactly the reverse. The patient is quite unconscious of the desire to pass water, and the bladder will gradually fill until it contains 40 to 60 ounces of fluid. If a catheter is not passed the urine will gradually accumulate until it begins to dribble past the sphincter from mechanical pressure, and the condition known as "dribbling from overflow" will have been established.

If, however, the distribution of the analgesia points to a local lesion above the ninth dorsal segment, a third condition of the bladder will appear. The urine will collect in the bladder until it has reached 15 to 20 ounces, and then will be passed without the patient's knowledge. Thus here the passage of urine is a normal but completely automatic act.

In the first class of cases the bed is always wet; in the second class the bed is never wetted if a catheter is passed about three times in the twenty-four hours; in the third class the bed is wetted three or four times only in the twenty-four hours, and is then saturated with a large quantity of urine.

Throughout the study of cases of nervous disease, evidence of the relation between pathological condition of certain viscera and sensory disturbances in the superficial structures of the body is constantly manifested. For instance, a man with caries of the spine suffered from a girdle sensation round the area of the eighth dorsal segment. At the same time he was greatly troubled by flatulent dyspepsia, which was untouched by drugs. It was, however, greatly relieved by counter irritation applied to the maximum tender point of the eighth dorsal area in the eighth space and mid axillary line.

Again in a case of tabes with analgesia over the area supplied by the tenth dorsal root, I saw an attack of complete paralysis of the small intestines simulating intestinal obstruction.

Thus, in conclusion, I feel inclined to think that the sensory supply of the viscera agrees with Dr. Gaskell's scheme for their motor and inhibitory supply in the following points:—

[1st]. The sensory fibres enter the central nervous system in three great groups. The highest of these is in the head and neck, the middle group lies beween the 1st dorsal and 1st lumbar segments of the spinal cord; the lowest group extends from the 5th lumbar to the 4th sacral segments.

[2nd]. Thus there are two gaps in the spinal cord which are not in connection with sensory fibres from the viscera. The higher of these consists of the 5th, 6th, 7th and 8th cervical segments, whilst the lower corresponds to the 2nd, 3rd and 4th lumbar segments.

CHAPTER V.

ON "GENERALISATION" AND GENERAL DISEASES.

So far, I have spoken as if disturbances in the activity of one organ produced pain and tenderness over certain definite areas only. If the human organism is otherwise perfectly healthy, this is so; but after the pain has remained localised for a considerable time, the condition of the central nervous system becomes profoundly altered. I pointed out above that in biliary colic, the tenderness over the eighth dorsal area generally makes its appearance on the left side as well as on the right. When one testicle is affected, the tenderness also tends to be bilateral, and the same applies to the kidneys, especially if the disease is a long and wearing one. Thus, there is a great tendency for both pain and tenderness to spread to the opposite side at the same spinal level. In all these cases it might be suggested that the organ receives most of its fibres from one side, but also a certain number from the other side of the cord; and there is probably much truth in this supposition.

But there is another and much more important form of diffusion or spreading. If I have an aching tooth, the pain is at first localised to the tooth affected. The longer the toothache continues, the more I become worn out, and the pain is rapidly accentuated by a "neuralgia," *i.e.*, that is a pain in the face. This neuralgia is soon accompanied by distinct cutaneous tenderness over a definite area on the face corresponding to the tooth affected. If I am anæmic, or if the pain remains untreated until my bodily health is affected, I no longer have a localised area of tenderness, but the pain and with it the tenderness spreads until the whole of one-half of the head and even the neck may be intensely tender. Thus at last the pain of an aching tooth has produced tenderness over areas which bear no relation to the affected organ.

But there is another method by which the pain caused by one organ may be ultimately accompanied by pain over an area corresponding to another organ. Certain viscera such as the uterus and breast are naturally closely related to another. Thus suppose pain and tenderness to exist in nsequence of the disease of one of these organs, the next rea that will be affected when this pain and tenderness spreads, is that belonging to the associated organ. Thus within twelve hours after labour, it is not uncommon to find a woman suffering from a pain under the heart associated with cutaneous tenderness which comes and goes with the "after pains." This pain under the heart may be increased by ergot, or by manipulating the uterus.

With orchitis or prostatitis, we also occasionally find that the patient complains of a pain at the epigastrium, representing the stomach area. This is put down to "hypochondriasis," and if it occurs in a woman as a consequence of ovaritis, she is said to be "hysterical." But this phenomenon is no more "hysterical," whatever that may mean, than is the reference of the pain and tenderness of an aching tooth to the back of the head or the shoulder.

This is the phenomenon which I have been accustomed to call "generalisation" of visceral pain and tenderness, and is of such common occurrence as to form a very important factor in the clinical picture of many diseases.

The order in which generalisation takes place, leads one to speak of the relative "specific resistance" of the centres for the sensory impulses from various organs. No very definite rule can be laid down to govern every case, but each case must be considered on its merits. However, the area which appears most easily in a woman, as a secondary affection, is the tenth dorsal; then perhaps the sixth dorsal, or inframammary, and then the various gastric areas, beginning first with the ensiform or seventh dorsal. In a man, the tenth dorsal appears rather less readily, whilst the ensiform appears with great ease. The cervical areas appear with ease in both sexes. On the other hand, the third and fourth dorsal associated with the lung, rarely appear as secondary affections. Thus the central connections of these areas in the cord appear to have a different specific resistance; some of them appear with ease even when the organs with which they are primarily associated are not affected, whilst others only appear after every other area in the body has become tender.

Now the greatest specific resistance seems to be posses by the areas of the two gaps; and when the gaps beco affected, it is only by a gradual creeping onward of th neighbouring areas. Thus, if we watch the genesis of universal tenderness in the leg, we find that first of all the twelfth dorsal and first lumbar in front, and the first, second, sacral and fifth lumbar of the posterior group are all tender. Then from the first lumbar above, the tenderness creeps over the second lumbar, whilst a similar process is occurring in the fourth lumbar below. At first, only the tender points appear, and then the areas gradually develop until the gap is filled up, and the whole leg is now tender.

In this connection I may say a few words on the difficult subject of dysmenorrhœa. A certain number of cases of dysmenorrhœa are entirely free from pain and tenderness in the inter-menstrual period, but develop definitely localised pain and tenderness, following the lines I have laid down, during menstruation. Of this class, the most satisfactory are those with some affection of the cervical canal, such as chronic cervical catarrh or polypi. They may be entirely free from pain until the flow comes on, when they develop pain and tenderness over one or more of the following areas: —the eleventh or twelfth dorsal, first lumbar, fourth, third, or second sacral. In some cases a considerable number of these areas are affected, but even at the height of the disturbance the tenderness does not transgress the limits of the areas supplying the pelvic viscera. Moreover, the areas corresponding to the part which is chronically diseased are always most affected.

With slight degrees of ovaritis, a second type of dysmenorrhœa makes its appearance. Here there is generally pain and some tenderness, especially after examination, over the tenth dorsal area, in the intermenstrual period. Just before menstruation begins, this tenderness becomes very intense, and by the time the flow is established all the areas which correspond to the pelvic organs are affected. Moreover, the areas of the gap may become tender by extension, until at last the whole body and leg, below the upper border of the tenth dorsal area, may be tender. In such cases, not only are the cutaneous (superficial) reflexes exaggerated, but the knee-jerk also becomes exaggerated. This is beautifully seen in those rare cases where the tenderness remains unilateral.

Now we pass to the third class of pain during menstruation in which the patient has some local affection which causes more or less constant visceral pain. This may be some error of refraction which causes its proper tenderness (which I shall describe and discuss in my next paper) over the middle of the upper lid and front of the scalp. It may be chronic cough, causing tenderness over the maxima of the second dorsal. It may be chronic gastric disturbance, causing pain over one of the gastric areas; or it may be chronic constipation, producing the well-known pains over the tenth and eleventh dorsal areas. Whatever form this pain normally assumes, it is enormously increased by the disturbing influence of menstruation on the nervous system. The hypermetrope will suffer from agonising headache, which may spread on to the neck. The patient with the cough suffers from pains and tenderness in the chest and arms, and in the lower branches of the cervical plexus. The patient with gastric disturbance has tenderness over more or less every gastric area. Moreover, there may be two centres of spread, which gradually widen until they meet, and thus there may be hardly a part of the body that is not tender. And now the patient is put down as "neurotic" or "hysterical," and all thought of rational treatment abandoned.

But as I shall show below the same phenomenon appears in even the most stolid of us males under the influence of a rise of temperature. Anyone who has suffered from a bad tonsillitis with fever, knows to a certain extent the symptoms of dysmenorrhœa, with its pain, aching and tenderness in the loins and back.

Thus the changes in the nervous system accompanying menstruation must be put down as one of the most potent causes of "generalisation" of visceral pain and tenderness.

Again, as I pointed out above (p. 92) a normal labour is frequently followed by some pain and cutaneous tenderness.

But the pain and tenderness very rarely overstep the areas associated with the pelvic viscera. But after abortion "generalisation" is very common, and although the course of the puerperium is normal, the patient will frequently complain of pain and tenderness elsewhere than over areas representing the pelvic viscera. It would appear as if the resistance of the nervous system were increased *pari passu* with the progress of normal pregnancy to bear the strain of normal labour; but that when abortion takes place it is unprepared for the strain put upon it and generalisation takes place easily.

Anæmia is one of the commonest causes of such generalisation. A patient may be anæmic and yet suffer from no pain or tenderness. But as soon as some cause for referred pain is present generalisation at once begins. Thus if an anæmic women menstruates she not only has pain over the tenth dorsal area corresponding to the ovary, but she has pain and tenderness over the areas of the sixth dorsal (heart), seventh dorsal (stomach), occipital and midorbital (eyes, etc.) If an anæmic man or woman have a toothache or suffer from hypermetropia they may have the most widespread tenderness and pain. And yet if the cause is removed this pain does not cease, at any rate immediately, for the resistance of the centre being once broken down is with difficulty recovered. In every such case of anæmia I believe the starting point for generalisation will be found in the affection of some organ. Where widespread tenderness of the head and neck are present and headache is a marked feature, some error of refraction is often present. In some cases the starting point of the pain is in the cough of some lung or bronchial affection. A very large number start originally in the pain of chronic dyspepsia, and an even larger number in the pain caused by some irregularity in the pelvic viscera.

Thus it is not sufficient simply to treat the anæmia or to treat the original cause and point of primary manifestation of pain, but both must be corrected.

But anæmia is not the only agent which will so lower the specific resistance of the central nervous system that a local pain will generalize. A sudden rise of temperature will produce the same effect. Follicular tonsillitis or any parenchymatous affection of the tonsils is associated with cutaneous tenderness, extending round the neck from the lower part of the mastoid behind, almost to the middle line of the neck just above the pomum adami. If one tonsil only is affected one side of the neck alone is tender over this area. But let the onset be sudden and associated with a rise of temperature to 101° or 102° F. We shall now find the same zone of tenderness, but in addition there will be pain and tenderness over the forehead, in the occipital region, over the maximum of the tenth dorsal area in the back, and at the epigastrium (representing the seventh dorsal), and probably also at several other points in the body and limbs, all of which represent the maxima of their various segmental areas. Thus a rise of temperature has added, to a simple area of tenderness representing the organ primarily affected, a multiplicity of painful and tender spots representing areas which could in no way be effected by the local manifestation of the disease.

I have attempted to show how a sudden rise of temperature may convert a simple patch of referred tenderness into a wide spread affection by breaking down the resistance in the central nervous system, which normally resist generalisation. And in some cases a sudden rise of temperature will produce this wide spread tenderness without our being able to recognise any area of primary disturbance. This condition is produced most easily by true epidemic influenza. In this disease the patient notoriously complains of "pains all over," and careful examination shows that these pains are associated with tenderness in the skin. A glance at fig. 38 is sufficient to reveal the resemblance of these points of cutaneous tenderness to the "maxima" of which I have spoken so frequently in this paper. In those cases of influenza which have their greatest incidence on the lungs, the most prominent spots are situated over those areas which usually appear in pulmonary disturbances. In the intestinal group, on the other hand, the maximum disturbance falls over the spots related to the intestinal tract. In some cases of influenza where the local manifestations in any one organ were very marked, I have actually seen whole spinal areas marked out by tenderness. In every case I have selected male cases and only such as had a well marked elevation of temperature, making the diagnosis a certainty. Moreover, I have only taken cases which occurred during the last epidemic, and have not included sporadic cases occurring at other times of the year.

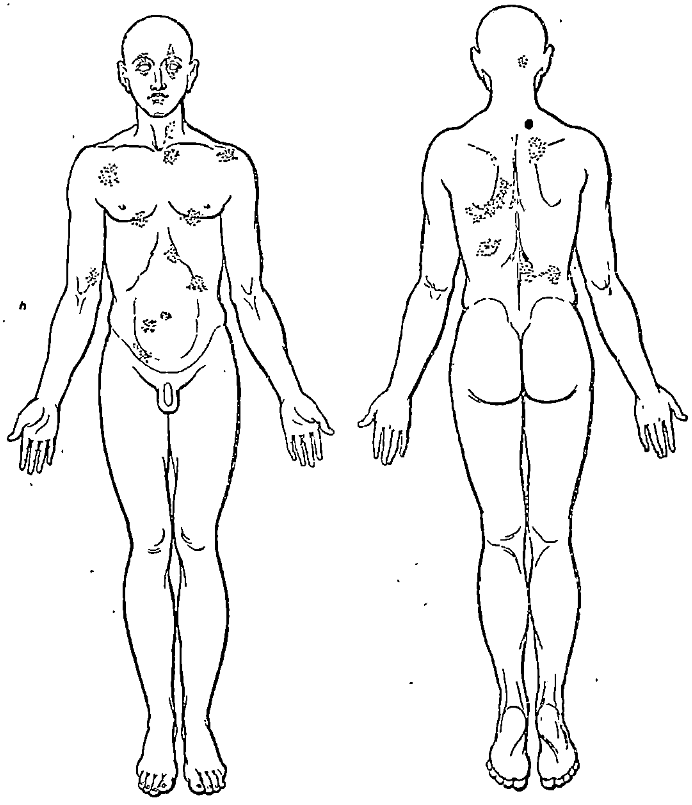

FIG. 38.

On the right hand side of the body in each figure are shown the areas of cutaneous tenderness during an attack of influenza in Case No. 50.

On the left hand side of the body are shown the areas of cutaneous tenderness which appeared during an attack of influenza in Case No. 51.

Case 50.—Robert F., aged 35. U. C. H., January 4, 1892.

January 1. Cold shivers, headache, pains all over especially in the joints.

January 4. Temperature 100° F.

Great cutaneous tenderness over the areas on the right hand side of fig. 38. His eyes ache and water, and the whole upper part of the body feels as if it had been "bashed."

Case 51.—Frank B., aged 16. U. C. H., January 6, 1892.

Typical influenza. Temperature 103°. Pains in head, eyes and upper part of body.

Marked cutaneous tenderness as on left hand side of fig. 38.

But influenza is not the only specific disease which produces these tender areas; they are sometimes present in typhoid fever. The most marked case I have ever seen was in my own brother in whom the temperature ran up very rapidily reaching 102° on the second day and 104° on the third day. He was very bright in spite of the high temperature and was able to give me a brilliant description of the locality of his pains and the peculiar "beaten" feeling he had over certain spots of his body. Fig. 39 shows the tender areas on the third day. As he lay in bed he tested his own body and without my suggestion found the greater number of the tender areas himself and marked them on his body for me. Such a condition is however uncommon in typhoid, for the temperature does not run up as a rule rapidly enough to produce this wide spread tenderness. The usual condition of a patient suffering from typhoid in the first week is a band of tenderness more or less well marked over the area of the tenth or eleventh dorsal segments with spots over the forehead corresponding to areas I shall discuss in my next paper. I have however seen one case besides the one just mentioned in which the tenderness was fairly wide spread and in this case also the temperature ran up rather rapidly.

Case 52.—Christopher H., aged 22.

Seized with frontal headache on night of December 22, 1891.

On evening of December 23, temp. 102°. He complained of headache over the eyes and pain in the right shoulder when he worked his Indian clubs. He also had pain in the lumbar region when he stooped..

Dec. 24. Temp. reached 104°. Cutaneous tenderness over his forehead associated with frontal headache. Cutaneous tenderness over the abdomen and loins marking out the tenth dorsal spinal area. The tenderness is so marked that he called a touch with the blunt end of the pin "a prick."

Dec. 25. Temp. now varies between 102° and 103.8°. The tender band in the abdomen and loin is no longer so well marked, but the whole body and head are dotted over with points of cutaneous tenderness. He himself found cutaneous tenderness over the following points and drew my attention to them. Tip of ninth rib, upper and outer part of thigh, angle of scapula, abdomen and loin, and in the spot on the calf. He said sometimes his back was worse, sometimes his calf, and sometimes his head.

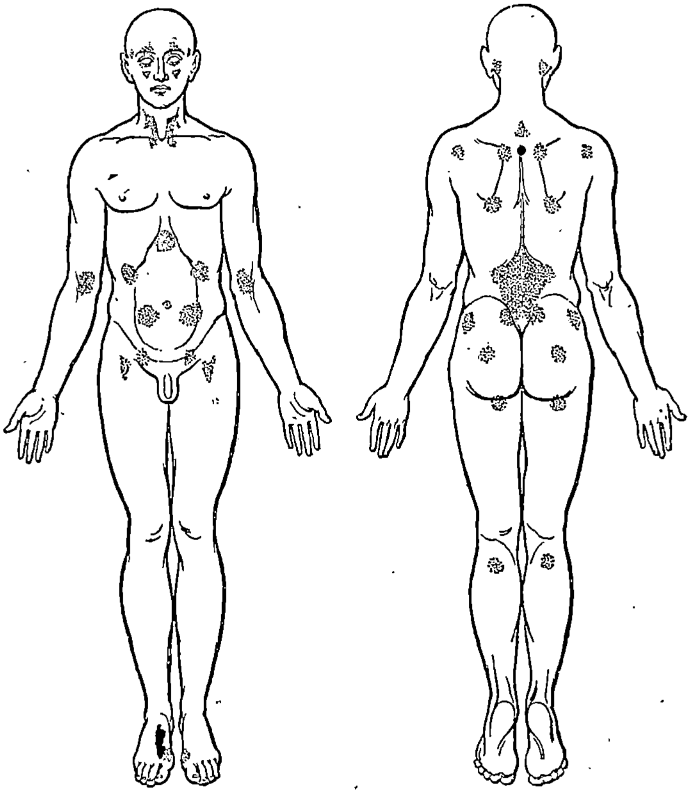

FIG. 39.

To show the areas of cutaneous tenderness which appeared in a case of typhoid in which the temperature ran up rapidly (Case No. 52).

Dec. 28. All cutaneous tenderness has disappeared. The temperature now varies between 102.4° and 103.6°. He has one stool daily and vomits slightly between midnight and 3 a.m.

Dec. 31. Typical diarrhœa set in and he is now beginning to get some cutaneous tenderness over the ninth and tenth spinal areas. The spleen can just be felt.

Jan. 9. The temperature is beginning to fall. Diarrhœa is ceasing.

Jan. 16. Temp. has not risen above normal. Uninterrupted convalescence without complication.

Thus this case showed a typical attack of severe uncomplicated typhoid differing from those seen every day in the rapid rise of temperature and in the fact that observations were made from the second day of the illness.

In acute rheumatism in the very early stages before the joints begin to swell definitely, similar cutaneous tenderness may also appear, though seldom, and always to a less extent than in the diseases I have mentioned so far. I saw one case of a man in whom the cutaneous tenderness was so marked that he was sent into the ward as a case of "hysterical" disease with rise of temperature. The next morning the joints had swollen and the cutaneous tenderness in the "ovarian" and "supra-orbital" regions had gone.

In one case of smallpox which I was fortunate enough to see in the stage of petechial rash not only was there tenderness (*vide* fig. 40) but the rash marked out certain areas in a beautiful manner; and I then saw that the so-called femoral triangles which are said to be so characteristic of this rash are in reality a rash marking out the thigh flaps of the twelfth dorsal spinal area. However the distribution of rashes from the point of view of the areas I have described in this paper are too big a subject to treat here.

Case 53.—John M. aged 18. U.C.H.

Has been ill nearly 8 days. The typical variola rash is beginning to appear irregularly all over his body. The early petechial rash is still present over the areas in fig. 40.

He looks very ill and complains of pain in the back (lumbar region) and in the abdomen. Definite cutaneous tenderness (head of pin called "prick") over the areas shown on the left hand side of fig. 40.

As we are discussing fevers it is interesting to notice that the drugs which are so much used to relieve referred and nervous pains are the antipyretics, such as antipyrin, antifebrin, phenacetin, &c.

In all the cases of generalisation which we have considered thus far the cause of the diminished resistance in the nervous system was a definite and recognisable one, such as anæmia, chronic pain or pyrexia. In the next class of cases the diminution in resistance is produced by some change in the nervous system itself. This primary central break down of resistance may be an inherent defect in the nervous system of the individual or it may be produced by some psychical shock.

Thus in cases of salpingitis and dysmenorrhœa it is not at all uncommon for the pain and cutaneous tenderness to lie strictly over the areas which correspond to the organs affected until, under the influence of some mental trouble, a love affair or a death, this pain and tenderness will suddenly generalise extending to the arms, chest and head, and the case will be converted into one of "hysteria." As soon as the generalisation has taken place local measures cease to afford relief, and removal of the ovaries or tubes will only add fuel to the fire by superposing a second cause for decreased resistance.

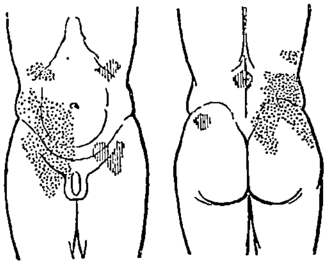

FIG. 40.

The right hand side of the body shows the distribution of the petectial rash in a case of small pox. Note the curious resemblance of the "femoral triangles," so characteristic of this rash, to the 12th dorsal area.

On the left hand side of the body is shown the cutaneous tenderness which accompanied this rash. This tenderness was associated with the well-known "aching in the back and groins."

The rash has been represented on the one side and the tenderness on the other for the sake of clearness.

This leads me to a consideration of the various forms of altered sensation which occur in "hysteria." I should feel inclined to divide them into two great groups.

1st. The cerebro-spinal.

2nd. The psychical type.

In the cerebro-spinal type I have been able to watch the gradual development of a case through the following stages.

(*a*) The stage in which there was well marked tenderness over areas corresponding to a diseased organ.

(*b*) Generalisation under the influence of long continued pain, anæmia or mental shock, so that the whole body becomes dotted over with areas of cutaneous tenderness.

(*c*) The cutaneous tenderness then passed into analgesia or loss of the sensation of pain with intact sense of touch.

(*d*) Touch also became affected and the greater part of the body became insensitive to pain, touch, heat or cold.

Now the characteristics of the cerebro-spinal type are as follows:—

(1) The sensory disturbance is always mainly one of pain, heat and cold, and the area affected corresponds to the areas of spinal segmentation at any rate in the early stages. (Later they may became altered by secondary suggestion). Fig. 41.

(2) The reflexes are always increased over the affected spots when the disturbance takes the form of hyperalgesia or lost when it takes the form of analgesia.

(3) There is as a rule no limitation of the visual field in the cerebro-spinal type.

On the other hand in the psychical type—

(1) Alterations follow natural lines,[1] *i.e.*, the loss of sensation runs round the limb limited above by the ankle joint, by the knee or by Poupart's ligament, loss of sensation on the abdomen occupies what is vulgarly called "the belly" or the "back," *i.e.*, it is limited by the costal margin and the lines of the iliac bones in front or by "the spine" behind. It takes "the eye" (without the forehead), "the mouth" (without the lips), "the chest" (without the arms). In fact the loss of sensation follows fundamentally different lines from that in the first class. Fig. 42.

(2) Sensation of touch, pain, heat and cold are absolutely coterminous, whereas in the first type pain is always lost before the sensation of touch and the two are lost to a very different extent.

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[1] These may be ultimately found to correspond to the cortical distribution of sensation.

(3) The reflexes are unaltered over the anæsthetic or analgesic areas. This I believe to be a very valuable distinction.

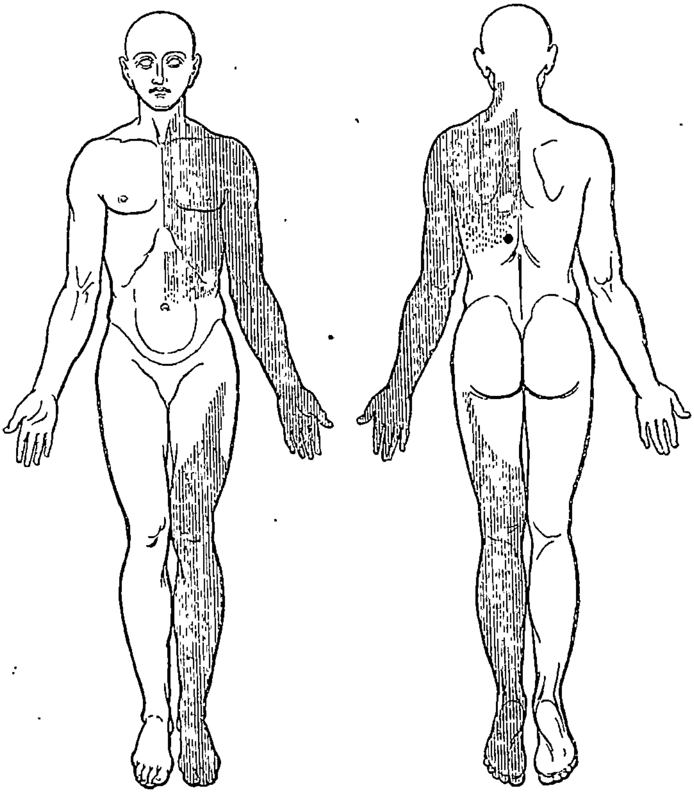


FIG. 41.

To show the distribution of analgesia in a case of "functional" disease (cerebro-spinal type) Case No. 54. All superficial reflexes to painful stimuli, and to hot and cold stimulation were lost over the areas in the figure.

The loss of sensation to touch (anæsthesia), was much less extensive, and was not co-terminous with that of pain, heat, and cold.

(4) There is almost always considerable limitation of the visual field in the psychical type (or hysteria proper).

Again the prognosis in the psychical type is much better than in the cerebro-spinal type.

Case 54.—Cissy P., aged 20, farm labourer. Queen Square (Dr. Buzzard), February, 1892.

August 1, 1891. Rose in the morning with pain in left side in mid-axilla and in front of chest under left breast; came as far forwards as base of sternum. "Like a knife" stabbing through from the front to the back, it made her cry out and feel faint. Went out to stack hurdles but was obliged to come home. Went to bed; hot skin; thirsty; no appetite.

August 4. Got up and went to work. Had to come home again on account of the pain. Went to bed and had a regular shivering fit, lasting about half an hour. Teeth chattered and shook all over. Then became very hot. Stopped at home, mostly in bed, for two weeks, and then went away for two weeks more. During the first two weeks "had an illness" (no details obtainable), but did not call in a doctor.

On returning to work in September felt the pain in the side again; but in twenty-four hours the pain disappeared, and she noticed that the left side was "numb." She could not hold the hurdle bands in her left hand and the left foot became numb and cold.

This gradually increased. In December began to walk with a stick.

Always healthy up till August, 1891. Has worked as a farm labourer for several years.

Menstruation began at sixteen. Every four weeks. Lasted five days. No pain. About November, 1891, it became irregular and has remained so ever since.

Condition on admission.—Medium sized girl. Not anæmic. Fairly intelligent. Bucolic stolidity. Sleeps well. No vomiting. No headache.

Stands well and does not stagger with the eyes shut. Drags left foot markedly as she walks. Left leg below the knee dragged like an inert mass. Grasp of right hand 40, left hand 30. All movements of left leg and foot well performed. Can dorsiflex and extend ankle.

All sense of position in left foot lost. Sense of position in left ankle impaired. Can recognise position of knee. Sense of locality in left hand perfect.

Sensation of touch impaired over the greater part of the left side of the body. Thus all sensation of touch is lost in the left leg below the knee, back and front; in the left hand, arm, shoulder and trunk, from a little above the angle of the jaw downwards, as far as a line running forwards from above the ninth dorsal vertebra to a little above the umbilicus. Neither upper nor lower limits are sharp lines, but gradually shade off. Moreover, the anæsthesia does not quite reach the middle line in front or behind. Below the umbilicus the sensation of touch is normal, but the upper and outer parts of the thigh are partially anæsthetic to touch.

The analgesia, or loss of painful sensation, is as in fig. 41 Sensation of heat and cold are lost over practically the same areas.

The superficial reflexes are absent on the left side, over the analgesic and anæsthetic areas. Thus the plantar reflex is abolished on the left side, to both touch and pain. Superficial reflexes are normal on the right side. Thus the reflexes are absent from the upper part of the abdomen on the left side, but present below the umbilicus.

Knee-jerks brisk both sides. Wrist tap present on both sides, but distinctly more ready on the right side than the left. Pharyngeal reflex present on both sides.

Vision normal. No diminution of the field of vision. No colour blindness. No ophthalmoscopic change.

Hearing normal right side. Watch heard only in contact left side.

Taste and smell good.

Loss of sensation to touch on left half of tongue.

Pain lost on a strip occupying the outer portion of the posterior part of the left half of the tongue.

No ocular paralysis. No nystagmus. Pupils equal; react to light and accommodation.

No pain or tenderness in the spine.

Urine, sp. gr. 1020; acid normal.

She rapidly improved, and on March 1, 1892, sensation was good all over body and limbs, and the superficial reflexes had returned. Discharged cured.

Case 55.—Samuel R., aged 13. Queen Square (Dr. Ramskill and Mr. Horsley).

January 6, 1892. Severe blow across left hip and lower part of spine. He went to school but was lame the same evening and the next day.

Two or three days later sharp stabbing pain came on in the lumbar region. It shot down the left leg.

Six days after the blow took to his bed on account of increasing pain. He said he could not move on account of the pain. When in bed he felt numb in the upper part of legs, thighs and hips. He had occipital headache and intolerance of light.

From January 6 to January 10 passed no urine. Also passed none from January 12 to January 16.

In April he was seen by Mr. Horsley, who sent him into Queen Square Hospital.

On admission.—Fairly well nourished boy of 13. Slight vertical headache. Fairly intelligent but lethargic. Dislikes the effort of taking part in the amusements of the ward. Could not at first be got to do the fancy work with which most of the patients amused themselves. Sleeps well, but at the onset of the illness used to dream he was falling almost every night.

Complains of slight pain in the back over the third and fourth lumber spines. Some tenderness on percussion but there is a well marked bruise in this position.

No vomiting.

He has some difficulty in raising himself in bed for he says it causes pain in the back. All movements can be performed by the lower limbs, but he complains of pain in the lower part of the back when he lifts his legs off the bed. Can stand and walk without help, but cannot walk far on account of the pain in the back.

Sensation to touch, pain, heat and cold are lost over the area marked in fig. 42. Yet in spite of this loss of sensation the cremasteric reflex can be beautifully obtained on both sides from the front of the thigh and the gluteal reflex from the posterior aspect. It is interesting to note that the cremasteric reflex can be obtained from the front of the thigh as far out as the level of the anterior border of the great trochanter. At this point we begin to change on to the sacral areas, and the gluteal reflex replaces the cremasteric. This experiment could be repeatedly performed in the patient in spite of the anæsthesia. Sensation over the rest of the body is normal.

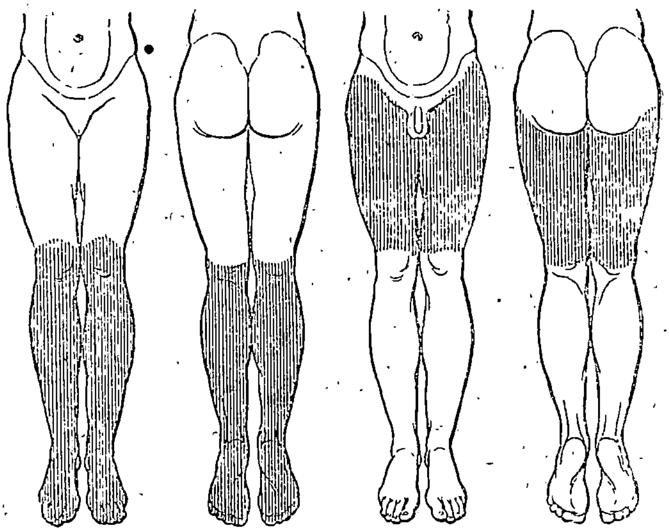


FIG. 42.

The figures on the left side represent the loss of sensation to touch, pain, heat, and cold in a "hysterical case," No. 56 (Psychical type).

The figures on the right side represent the loss of sensation to touch, pain, heat, and cold in a case of "traumatic hysteria," No. 55 (Psychical type).

Knee-jerk present and equal; not exaggerated. No ankle clonus. Plantar reflexes present. Wrist tap normal.

Smell good. Taste good. Hearing good.

Very marked limitation of the field of vision in both eyes.

No ophthalmoscopic changes.

No ocular paralysis. No nystagmus.

Pupils medium sized. React to light and accommodation.

All other cranial nerves good. Tongue protruded straight.

Appetite poor. Tongue clean. Tendency to constipation.

No physical sign in abdomen.

Heart sounds normal, no murmur.

Urine only passed two to three times a day. Quantity uncertain, sp. gr. 1020. Acid. No albumen.

He was placed on Weir Mitchell treatment.

Sensation varied a good deal. Sometimes the lower limit of the loss was at the knee. Sometimes at the ankle and sometimes only the sole of the foot was unaffected.

He increased in weight from 6st. 2¼lbs., to 6st. 8lbs., in seven weeks.

He was discharged cured October 10, 1892.

Case 56.—Mary G., nurse, single, aged 32. Queen Square (Dr. Tooth).

Summer of 1891, pain in upper lumbar region of the back.

October, 1891. Pain and tight feeling about lower part of chest. Six weeks afterwards began to vomit directly after food. She is said to have vomited blood, but never in large quantities. Was supposed to have gastric ulcer.

The vomiting continued two to three times a week until February, 1892, when she changed her doctor. The new doctor stopped the vomiting immediately.

She appears to have had influenza about Christmas, 1891, which left her with pain and tenderness in the spine, extending from the bottom of the back to the top of the head. This pain became gradually worse, and she began to slowly lose power in her legs. Was now treated for spinal caries.

No incontinence or retention of urine at any time.

Constipated. An enema has been administered three times a week ever since October, 1891.

Has always lived at home with her mother, and has never earned her living though she calls herself a nurse.

Is very liable to fainting. Very easily upset.

Menstruation regular every four weeks. Lasts three days. Not profuse. No marked pain.

Marked family history of phthisis, but not of neuroses.

Condition on admission, August 17, 1892:—

Medium sized woman of 32. Face pale. Lips somewhat pale. No œdema. Thin and ill-nourished. Temperature 98°, both in the morning and the evening.

She lies on her back complaining of pain in the spine and inability to move her legs. Quick to observe the occurrences in the ward. Is fond of reading. Memory fair. Answers questions well. Voice low. Speech normal. Occipital and vertical headache. No vomiting for several months.

She cannot stand, and cries bitterly when she is taken out of bed, and supported on her feet. She complains that it causes her pain in about the region of the second lumbar spine. She cannot dorsiflex or extend the foot to command. If, however, the foot is passively dorsiflexed all the anterior tibial group of muscles are seen to contract. Moreover, when her leg hangs out of bed the feet remain in the normal position and do not drop. She cannot extend the foot or move the toes to command, but the foot remains in the extended position even when opposed to gravity. She cannot extend the knee to command, but when the leg is lifted off the bed the knee remains rigidly extended. Again, if the thigh is flexed passively the leg does not drop until after a considerable interval. Grasp is practically non-existent under observation, but appears to be fairly good for ordinary purposes. She does not complain of paralysis of the arms, but it is very difficult to get her to perform any movement of the arms to command.

The legs are thin and the muscles flabby, but there is no marked wasting of any group of muscles.

Electrical reactions normal.

She complains of pain in the upper lumbar region. There is intense cutaneous tenderness all down the spine limited to an area about two-and-a-half inches in breadth. This merges above into an area of tenderness over the occiput.

Touch, pain, heat, and cold are not felt over the area in fig. 42. The limits of the loss of sensation are absolutely coterminous for all forms. Sensation of rest of body normal.

She seems to have lost all sense of position as high as, and including the knee. She does not seem to know when her muscles contract under the faradic current as high as the knee. She can bear the strongest faradic current without flinching over the anæsthetic area.

Knee jerk exaggerated on both tides. Wrist tap brisk both sides.

No ankle clonus.

Plantar reflexes present to touch, but slightly diminished. Fairly brisk to a pin prick. Thus reflexes can be obtained, even over the anæsthetic area.

Abdominal reflexes good.

Pharyngeal reflex absent on both sides.

In spite of the tenderness of the spine, the usual tests for bone disease give a negative result. No visible abnormality in the spinal column.

Taste, smell, and hearing good.

Myopic. No opthalmoscopic change in fundus or discs.

When tested with Snellen's test types, says she can only read $\frac{6}{200}$ [$\frac{2}{60}$]. Again, she reads Jäger 1 at the usual distance, but rapidly approximates the board to her face, saying it gets quickly dim. Finally, she only reads J. 10 badly. This occurs with each eye.

Marked diminution in the field of vision for white and black and to a still greater extent for colours.

No nystagmus. Cranial nerves normal, excepting that the sensation of the pharynx seems to be abolished.

Sphincters normal.

Circulatory and respiratory systems are normal.

Appetite poor, tongue clean. No enema was administered during her stay in the hospital, but she was told that her bowels would act normally. They acted regularly every day without artificial aid of any kind.

August 27. Feels the wire brush, and cries with the pain.

August 29. All forms of sensation now good. They returned simultaneously.

August 30. Can only bear wire brush at half the full strength.

September 1. Moves legs to command, though slowly.

September 8. Pharyngeal reflex has returned.

December 7. She left the hospital cured, excepting for occasional pain in the back.

These three cases exemplify the difference between the two types of so-called hysteria. For Case No. 54 was characterised by the following points:—

(1) The loss of sensation followed the lines laid down in this paper for the distribution of sensory disturbances in visceral disease.

(2) Reflexes were abolished over the anæsthetic area.

(3) There was no limitation of the field of vision.

On the other hand Cases No. 55 and No. 56 agreed in the following points:—

(1) The loss of sensation followed lines incompatible with the areas affected in visceral disease.

(2) The plantar reflexes in Case No. 56 and the cremasteric and gluteal reflexes in Case No. 55 were present over the anæsthetic area.

(3) There was marked diminution in the visual field.

Now we can see how it is possible for the cerebro-spinal type to become associated with organic disease. The question at present agitating neurologists is the relation of disseminated sclerosis to "functional" disease. An enormous number of cases of disseminated sclerosis with tremor nystagmus and optic atrophy, have been preceded by years of undoubted "hysterical" manifestations—alteration in sensibility, vomiting, double sciatica, &c. Moreover, many of them have in this "hysterical" stage been "cured" more than once.

Thus I should like to suggest that the term "hysterical" be kept for the psychical type only, whilst the term "functional" disease be made to include all forms of generalised pain or loss of sensation following definite cerebro-spinal lines, whether the alteration in the nervous system is caused by anæmia, pyrexia, menstruation, &c., or some truly nervous influence, such as shock, or long continued visceral pain.

CHAPTER VI.

THEORETICAL.

§ 1. Why is pain referred?
§ 2. Why is the skin tender in visceral disease?
§ 3. The central relations of the various forms of sensation.

§ 1. *Why is pain referred?*

This constant reference of pain to the skin when the lesion affects an internal organ at first sight seems inexplicable. But I think a consideration of the following facts brings us a little nearer the comprehension of its significance and of the method in which it originally came about.

If I prick my foot I do not feel pain anywhere except in the foot itself about the point pricked. If the various parts of the body are tested it is found that our powers of localisation for a painful stimulus are very fairly accurate even over such comparatively insensitive parts as the back. If however the power of perceiving pain is diminished over a certain area on my foot, as occurs so frequently in such diseases as tabes dorsalis, the pain is no longer localised to the point pricked but is referred higher up the leg or if the sensation of the opposite foot is good to the corresponding point on the opposite foot. This is a phenomenon known as allocheiria. This is still better shown in cases of nervous disease, where the patient has lost the sense of pain, to a considerable extent, over the areas of both sides of the abdomen corresponding to the distribution of the tenth and eleventh dorsal segments. If such a patient is pricked at a point about 2 inches below the umbilicus he will feel the pain but will refer it to a point about a similar distance above the umbilicus. A second prick one inch below the umbilicus is referred to a point about the same distance above. A third prick almost on the border line which separates the area of diminished sensation from the area over which sensation is normal will be referred to a point just on the healthy side of the line. Thus, although the actual stimuli have been ascending towards the umbilical line, the patient says that he has experienced a series of pricks descending towards the umbilicus. If he is now pricked three times on the healthy skin over the spots to which he originally referred his pain, he will say that the pricks are ascending. Thus six painful stimuli applied over the abdomen, beginning at a point about 2 inches below the umbilicus in the area of diminished sensation and ending two inches above the unbilicus in the area of healthy sensation, will be said to be three pricks descending towards the umbilical line followed by three over the same spots ascending from the umbilicus.

This rule applies to all forms of sensation. If the sensation of touch is affected, but pain and heat and cold are normal, touch only will be referred, whilst heat, cold and pain are rightly localised. In the same way if heat or cold only are affected whilst pain and touch are normal, heat or cold stimuli will be referred, whilst pain and touch are rightly localised. I have records of cases in which only one form of the four sensations was wrongly localised, whilst the others were localised over the point of stimulation.

In all such cases of wrong localisation the particular sensation which is wrongly localised must be diminished, and must not be absolutely lost over the affected area. If the power of perceiving this stimulus is totally lost the stimulus is simply not perceived and there is no allocheiria.

Now the localisation of sensation is not a physical but a psychical phenomenon. It is not a question of the number of rods and cones or of the number of nerve end organs, but is a phenomenon of association. Thus supposing a prick was so applied that it just stimulated one nerve end organ, this stimulus so caused would pass up one fibre and probably remain distinct all up the cord. But in consciousness the final result of this stimulus appears as a disturbance in the psychical field over a wide area with its maximum at one point representing the psychical unit which corresponded to the part stimulated. The disturbance over this point is, however, so greatly in excess of that over any portion of the diffusion field, that the diffusion field goes under the threshold and the position of the stimulus is rightly localised. If, however, the presentation to consciousness, which is the psychical equivalent of the point of stimulation, is less powerful than usual, owing to the physiological diminution produced by the partial analgesia of the area stimulated, the diffusion field will no longer be driven under the threshold. Thus the psychical effect produced by the area of diffusion may be even in excess of that representing the point of stimulation, and the latter will now go under the threshold. Under such conditions the position of the stimulation will no longer be localised at the point stimulated, but somewhere in the area of diffusion.

Now whenever an area representing one or more spinal segments on one side of the body is partially anæsthetic, whilst the sensation of the areas representing the same segments on the other side is normal, a stimulus to the skin over the affected areas is referred to the corresponding point on the normal side. If, however, the segments on both sides are affected, the stimulus is referred to the area corresponding to the area next above or next below the affected segments in their origin from the cord. Thus supposing the first, second and third lumbar areas are partly analgesic, a prick over their distribution will be referred to the area supplied by the twelfth dorsal or fourth and fifth lumbar. Now, as can be seen from plates I. and II., the posterior limits of these three areas closely abut on the limits of the second, third and fourth sacral roots on the outer and posterior aspects of the thigh. And yet a prick near the posterior border of these lumbar areas is not referred a few inches further back on to the distribution of the sacral areas, but to some point in the groin or below the knee (twelfth dorsal or fourth and fifth lumbar), more than 12 inches distant. This statement appears to be true for pain, heat and cold, which do not overlap, but is I believe not true for touch, for the distribution of the touch areas overlaps considerably.

These facts have an obvious bearing on the reference of the pain produced by visceral disease. For the viscera are notoriously insensitive to touch and to the stimulus of a cut with a knife. Thus after gastrotomy the mucous membrane of the stomach can be tickled with a feather, or gently pinched with the forceps without the patient's knowledge. In liver operations it is customary to stitch the liver to the abdominal wall, as a preliminary to opening the organ when adhesions have formed. If the second stage of the operation is done without an anæsthetic the patient is quite unable to say when the knife is plunged into his liver, and after the whole is over and the drainage tube in place, will ask "when the operation is going to begin." The same statement applies to the second stage of an inguinal colotomy, in which the gut is opened by a clean cut of the knife. The only stimuli which seem to act on the viscera are tearing or rending in character, in fact pressure stimuli.

Thus, as our viscera are so notoriously insensitive, and as we have never had the opportunity of developing the sense of localisation in them, owing to their inaccesibility to touch, it is not to be wondered at that the maximum pain is not felt in the organ affected. They are thus as regards the localisation of sensation in exactly the position of the area on the skin whose sensation of pain was diminished in comparison with that of its neighbours. A painful stimulus to an internal organ is conducted to that segment of the cord from which its sensory nerves are given off. There it comes into close connection with the fibres for painful sensation from the surface of the body which also arose from the same segment. But the sensory and localising power of the surface of the body is enormously in excess of that of the viscera, and thus by what might be called a psychical error of judgment the diffusion area is accepted by consciousness, and the pain is referred on to the surface of the body instead of on to the organ actually affected. I do not mean to state that pain is never referred to the organ affected. Far from it. The pain is frequently felt in the organ itself, but it is "dull," "heavy," "wearing," and not "sharp," "aching," "stabbing," like the referred pain.

Thus I believe that the phenomena of allocheiria and of referred pain in visceral disease are in nature and explanation essentially the same. Both depend for their appearance on the law that where a painful stimulus is applied to a part of low sensibility in close central connection with a part of much greater sensibility the pain produced is felt in the part of higher sensibility rather than in the part of lower sensibility to which the stimulus was actually applied.

§ 2. *Why is the Skin Tender in consequence of Visceral Disease?*

If impulses pass up the sensory nerves from an organ which is diseased to the cord they will set up a disturbance in the segment of the cord to which they are conducted. Now any second sensory impulse from another part conducted to the same segment of the cord will be profoundly altered. Under normal circumstances it would have set up its own proper disturbance in the grey matter and this disturbance would have been conducted to the brain. But now it no longer falls into a normal and quiescent cord but into one whose activity is already disturbed. The resultant stimulus conducted upwards is therefore a very different one from that which would have passed on under normal circumstances. In many cases the second stimulus will be exaggerated like rays passing to the eye through a convex glass. Thus if any segment of the cord is disturbed by painful stimuli from an internal organ a stimulus applied to the skin over the areas supplied by the nerve roots belonging to this segment will be exaggerated and a stimulus which normally was perhaps only uncomfortable would now appear to be very painful.

But in order that this condition may result the central connection between the sensory nerves from the viscera must be closely connected with that part of the cord into which the sensory stimuli from the skin are conducted. Now we never find cutaneous *hyperæsthesia* or increased sensitiveness to touch as the result of visceral pain but only increased sensitiveness to pain or *hyperalgesia*. It might be objected that we do not know of the possibility of hyperæsthesia apart from hyperalgesia but the following case illustrates the difference well.

A tabetic who is now under observation has complete loss of the sensation of pain over the thorax and abdomen. Thus he is quite unable to feel the prick of a sharp needle, and no reflex is produced by a sharp prick on the abdomen. But the slightest touch produces the most profound disturbance; an enormous reflex is produced and he says that the touch causes a feeling of the most exaggerated tickling. He complains that nothing he has ever experienced was equal to the exaggerated feeling of "ticklishness" which is produced by a light touch over the affected area. Thus the man has over the same area hyperæsthesia or exaggerated sensation of touch with analgesia or absent sensation to pain.

In the same way I have seen cases where even the mildest heat was said to "burn," though touch, pain and cold were normal; in such cases the reflex produced by the warmth was exaggerated whilst the reflexes produced by the other forms of stimulation were normal.

Now visceral pain always produces exaggeration of the sense of pain and sometimes exaggeration of the sense of heat and cold in the skin but never so far as my experience goes does it produce exaggeration of the sense of touch or hyperæsthesia proper.

§3. *The Central Relation of the Various Forms of Sensation.*

This leads me to another and wide-reaching question—What is the relation in the central nervous system of the various forms of sensation?

We have already seen reason to believe that the cutaneous distribution of touch overlaps considerably. On the other hand the cutaneous distribution of pain, heat and cold do not overlap appreciably. In this their distribution agrees with that of the trophic nerves to the skin from each segment of the cord, which, as I showed by a study of the distribution of herpes zoster, are also found not to overlap.

Then again, we find that visceral disturbances may cause exaggeration of the cutaneous sensibility to pain, to heat and to cold, and are also closely related to trophic disturbances on the skin, such as herpes zoster. On the other hand they never cause exaggeration of the sensibility to touch.

Now in central gliomatosis, usually known under the name of syringomyelia, pain, heat and cold are profoundly disturbed, and there is a particular proneness to the development of cutaneous trophic lesions. On the other hand touch is either not affected at all or affected very late in the disease when the growth has produced profound pressure effects. Thus here again we find that a lesion around the central canal of the spinal cord tends to destroy the one group and to leave the other unaffected.

Thus I think that these facts tend to show that there is an intimate connection between the central connections for the sensory nerves of the viscera and the nerves which supply the sensation of pain, heat and cold, and also those which exert a trophic influence on the skin. I do not therefore think we shall be far wrong in assuming that the trophic nerves to the viscera bear a similar relation to the sensory sympathetic fibres as the trophic nerves to the skin bear to the fibres for pain. If this be so it will help to explain the effect produced by counter-irritation of the skin on the activity of the viscera. Conversely it will also explain the occasional improvement in pains of a purely nervous origin by some visceral irritation, *e.g.*, the removal of lightning pains over the 1st, 2nd, 3rd and 4th sacral [rectum] areas in a tabetic by the administration of a purge such as castor oil.

Thus to sum up I think we may conclude that the central connections of the pain fibres from the skin and viscera are closely connected with one another. The central connections of the nerves for heat and cold, and for trophic disturbances in the skin must also be in somewhat close association, though probably not actually connected. On the other hand the nerves for touch from the skin (we do not know whether nerves for touch exist in the viscera) are widely separated centrally from those of pain. As to their paths in the cord I do not yet feel qualified to speak definitely.

It is now my pleasing duty to thank those who have been so liberal in their help during this investigation. Firstly, my best thanks are due to the physicians and surgeons of University College Hospital for the kind way they have permitted me to use their cases. I also have to return my sincere thanks to the medical and surgical staff of the National Hospital for Paralysed and Epileptic, Queen Square, and especially to Dr. Buzzard, my chief, for their generosity towards me. In each report from these two hospitals I have given the name of the physician and surgeon to whose kindness I am indebted for the case. In all cases the disturbance of sensation was worked out by myself, but in the surgical cases I am indebted to Mr. G. B. M. White, Surgical Registrar to University College Hospital, for the report of the patients' physical condition. I beg to return him my best thanks. My thanks are also due to Dr. Bowman and Dr. Wood, House Physicians at Queen Square, and to the House Physicians and Surgeons of University College Hospital for much kind thought and help in this laborious investigation.

PLATE I.

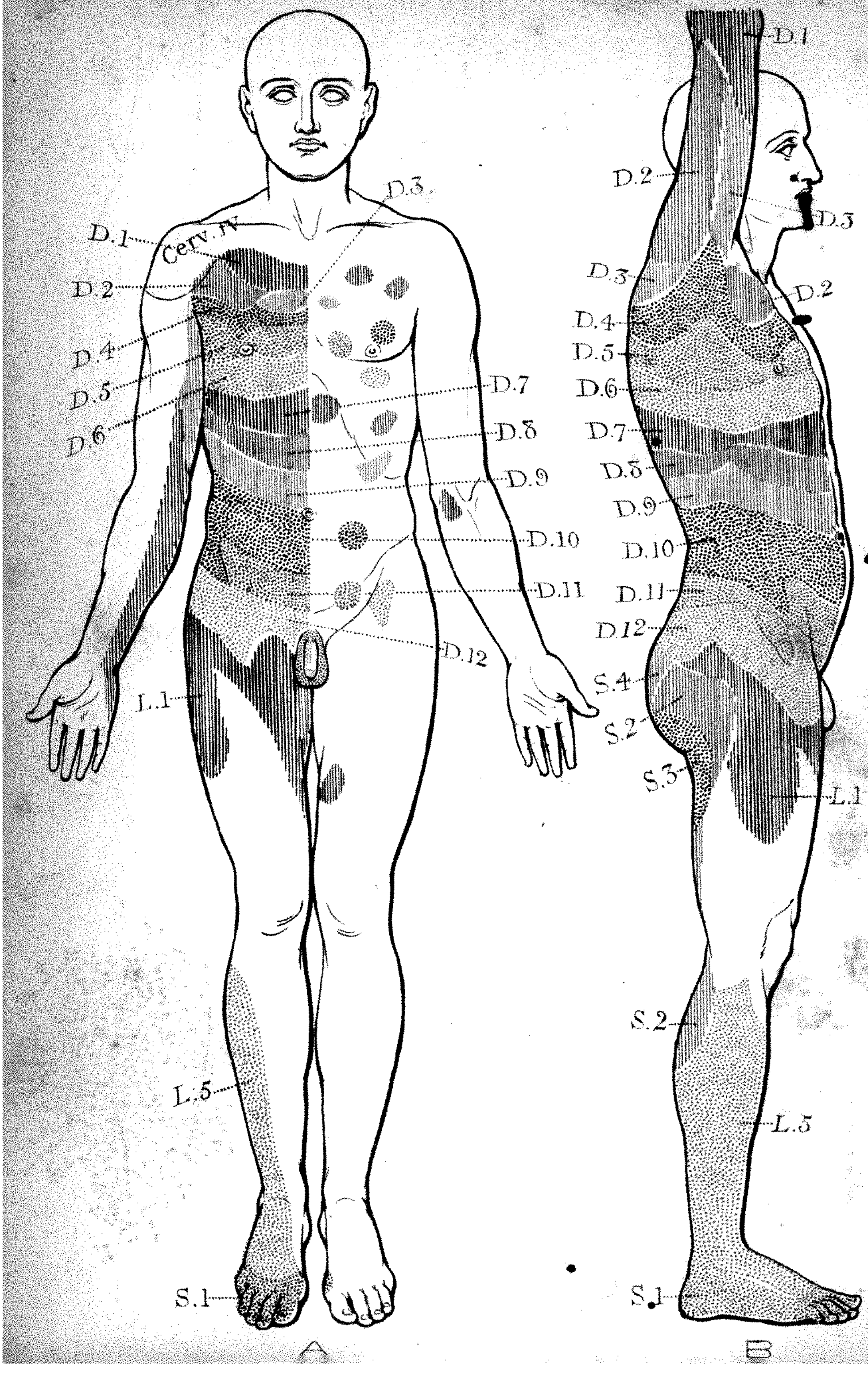
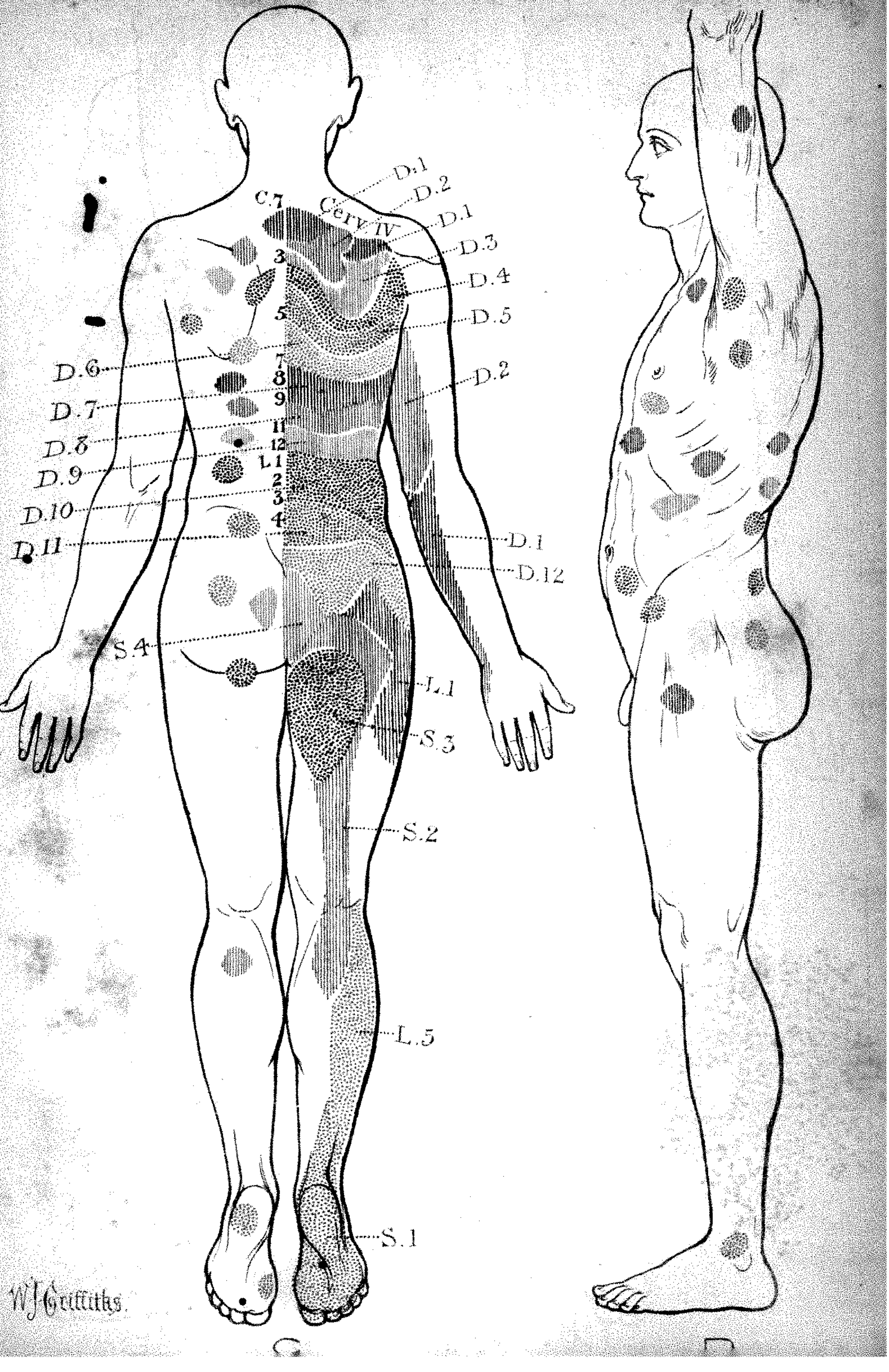

D.1
Cerv. IV
D.2
D.3.
D.4
D.5
D.6
D.7
D.8
D.9
D.10
D.11
D.12
L.1
L.5
S.1
A
D.1
D.2
D.3
D.3
D.2
D.4
D.5
D.6
D.7
D.8
D.9
D.10
D.11
D.12
S.4
S.2
S.3
L.1
S.2
L.5
S.1
B

PLATE. II.


D.1
D.2
C.7
Cerv. IV
D.1
D.3
D.4
D.5
3
5
7
8
9
11
12
L 1
2
3
4
D.6
D.7
D.8
D.9
D.10
D.11
D.2
D.1
D.12
S.4
L.1
S.3
S.2
L.5
S.1
W.J. Griffiths.
C
D

DESCRIPTION OF PLATES I. AND II.

These plates are intended to show the distribution of the segmental areas from the 1st dorsal to the 4th sacral. The form and extent of each area here figured is deduced from:

[1st]. The appearances presented by the cutaneous tenderness in visceral disease.

[2nd]. The distribution of the eruptions in 62 cases of herpes zoster.

[3rd]. The limits of analgesia (or loss of painful sensation) in organic disease of the spinal cord and roots.

I do not suppose that the areas here figured are absolutely correct, and can but hope that future investigation will show that their situation and extent bears a fairly close relation to the truth.

Cerv. IV. represents the lower border of the area supplied by the 3rd and 4th cervical areas.

Then follows a gap which is not usually represented in the disturbances of viscera. This gap corresponds to the 5th, 6th, 7th, and 8th cervical segments, and will be filled in, in my paper on the "Head and Neck."

D. 1 represents the dorso-ulnar or 1st dorsal area.
D. 2 represents the dorso-brachial or 2nd dorsal area.
D. 3 represents the scapulo-brachial or 3rd dorsal area.
D. 4 represents the dorso-axillary or 4th dorsal area.
D. 5 represents the scapulo-axillary or 5th dorsal area.
D. 6 represents the sub-scapulo inframammary or 6th dorsal area.
D. 7 represents the sub-scapulo ensiform or 7th dorsal area.
D. 8 represents the middle epigastric or 8th dorsal area.
D. 9 represents the supra-umbilical or 9th dorsal area.
D. 10 represents the sub-umbilical or 10th dorsal area.
D. 11 represents the sacro-iliac or 11th dorsal area.
D. 12 represents the sacro-femoral or 12th dorsal area.
L. 1 represents the gluteo-crural or 1st lumbar area.

Then follows a second gap which is not primarily represented in the disturbances of viscera. It corresponds to the 2nd, 3rd and 4th lumbar areas. A diagram showing this tract of skin split up into its segmental constituents will be found on page 53.

L. 5 represents the fibulo-dorsal or 5th lumbar area.
S. 1 represents the soleal or 1st sacral area.
S. 2 represents the sciatic or 2nd sacral area.
S. 3 represents the gluteo-pudendal or 3rd sacral area.
S. 4 represents the coccygeal or 4th sacral area.

PLATE I.

A. On the left-hand side of this figure the areas are shown from the front in their entirety. On the right-hand side the maximum spots of each area are figured, and in each case the colour is the same as that of the area to which each spot belongs.

B. This figure gives a lateral view of the areas marked in full on the left-hand side of fig. A.

PLATE II.

C. On the right-hand side of this figure the areas are shown from the back in their entirety. On the left-hand side the maximum spots of each area on the posterior aspect of the body are represented.

D. Shows a lateral view of the body on which the maximum spots of each area are marked as before.

To facilitate the recognition of these areas in any given case, I give a table on which are marked the viscera that are associated with tenderness in the various areas shown on these figures.

	HEART.	LUNG.	STOMACH.	INTESTINE.	RECTUM.	LIVER and GALL BLADDER.	KIDNEY and URETER.	BLADDER [Mucous Membrane and Neck].	BLADDER [Over distension and ineffectual contractions].	PROSTATE.	EPIDIDYMIS.	TESTIS.	OVARY.	APPENDAGES.	UTERUS [in Contraction].	UTERUS [Lower Segment and Os Internum].
D 1	X	X														
D 2	X	X														
D 3	X	X														
D 4		X														
D 5		X														
D 6			X			X?										
D 7			X			X										
D 8			X			X										
D 9			X	X		X										
D 10				X		X	X			X		X	X		X	
D 11				X			X		X	X	X			X	X	
D 12				X			X		X	X?	X			X	X	
L 1							X		X		X			X	X	
L 5										X						
S 1								X?		X						X?
S 2					X			X		X						X
S 3					X			X		X						X
S 4					X			X								X

THE NERVE CELL CONSIDERED AS THE BASIS OF NEUROLOGY.[1]

BY E. A. SCHÄFER, F.R.S.

It is obvious that the nerve-cell must always form the basis of the science of Neurology in all its branches. But although this cell has been studied for a great number of years it is only now that we are beginning to arrive at a definite understanding regarding its structure, functions and relation to other nerve-cells. This has seemed to me therefore a suitable occasion for gathering up such threads of knowledge as we at present possess, and laying them before the members of this Society.

It is consequent upon the application of new anatomical and experimental methods that our knowledge of nerve-cells has lately become extended. Amongst anatomical methods which have yielded of late the most valuable results are the Wallerian method in its latest application by Marchi (23); the method of staining *intra vitam* by methyl blue which was introduced by Ehrlich (4); and the method of staining with nitrate of silver after treatment with chromic acid and its salts which was introduced by Golgi, and which has yielded invaluable results, not only in the domain of the nervous system, but in every other branch of histology. Amongst recent physiological methods the investigation by the galvanometer and electrometer of the paths taken by action-currents has in the hands of Gotch and Horsley (13) given results of great interest.

We may most naturally begin with the *structure* of the nerve-cell. I do not know why one should restrict the term "nerve-cell" to the body of the cell and thus exclude from that term the cell-processes. This is not done for any other kind of cell, and it appears to me that the custom which has hitherto prevailed with regard to nerve-cells in this matter is not only inadvisable but even misleading. Waldeyer (45) has used the term "neuron" in this way to denote the whole nerve-cell, including all its processes. It is better, however, and simpler, to include under the term "cell," as is done with every cell of the body, the processes as well as the body of the cell. It will therefore be understood that by the term "nerve-cell" I shall denote not only, as has hitherto for the most part been the case, the body of the cell or the part immediately enclosing the nucleus, but all the several processes of the cell, and in that sense the word "nerve-cell" will be here used as identical with Waldeyer's term "neuron." The term "neuron" is short and convenient, but I shall employ it in a different sense, as will hereafter be seen, and I adopt this course the more readily because it has not yet come into very general use in Waldeyer's sense, and it is, I think, undesirable that it should do so.

---

[1] Presidential Address delivered at the Annual Meeting of the Neurological Society, January 19th, 1893.

The nucleus of the nerve-cell is noteworthy as compared with the nucleus of other cells by reason of its size, and it is large in proportion to the cell, *i.e.*, not only absolutely but relatively. If we examine a number of nerve-cells we find that the size of the nucleus and of the cell-body often bear a direct relation to the length of the processes which leave the cell. This is the case with the motor cells of the spinal cord and with the large projection-cells which are found in that part of the cortex of the brain from which the fibres of the pyramidal tract originate; it is also true for the ganglion-cells of the retina and the cells of the spinal ganglia. But I am bound to add that it does not appear to be an invariable rule, at least I doubt if it will apply to the visceral and vascular nerves. With regard to the structure of the nucleus the most important point in connection with this is the prominence of the nucleolus. This has long been noticed; in fact, one of the characteristics of a nerve-cell given in text books is that it contains a large and distinct nucleolus. There is also an intranuclear network as in most other cells which are not dividing, but this network is less distinct and more difficult to demonstrate than in many other cells. As for the protoplasm of the cell-body there is not much that is new to remark. The usual reticular appearance has been described in this as in the protoplasm of other cells (21, 25); and it is also well known that the fibrils which can be traced in the processes of the cell can also be traced into and even through the protoplasm. We may note also a point which distinguishes these cells from many others, the frequency, namely, with which we find pigment accumulated in the body of the nerve-cell. This fact has sometimes been interpreted to imply decadence of function (8); but it seems to me that it is usually to be interpreted as a sign of activity rather than of decadence. For there is no doubt that in other organs the presence of pigment in cells is accompanied by marked protoplasmic activity, which may be both chemical and physical. I need only mention in this connection the hepatic cells, the pigment-cells in the skin of the frog and the hexagonal-cells which form the outermost layer of the retina of the eye. And it appears to me that the relative abundance of pigment in adult human nerve-cells as compared with nerve-cells of the child and of the lower animals is rather an indication of functional activity than the reverse.

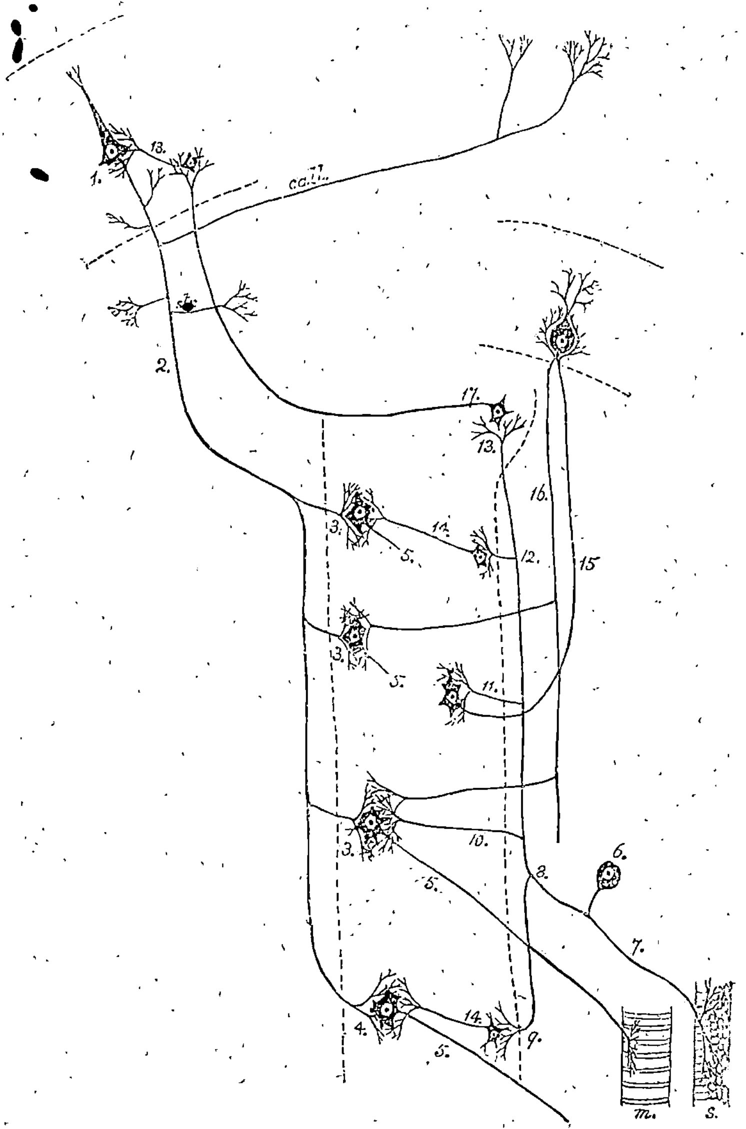
We may next consider the processes of the cell. Every nerve-cell has one or more; it is an absolute characteristic of nerve-cells to possess processes. The processes are of two kinds. The first and the only essential kind is that which has long been known as the axis-cylinder or nerve-fibre process (Deiters). It is also the first kind to show itself in the course of development of the nerve-cell (14). The other kind is that which was distinguished by Deiters as the protoplasmic process. This is not so essential, for we find many nerve-cells entirely destitute of it. A word is here again necessary with regard to nomenclature. The above terms have been long in use to indicate these two kinds of processes, but they are obviously inconvenient from their length and from the assumptions which they appear to imply. I propose therefore to term the axis-cylinder or nerve-fibre processes *neurons*; and the protoplasmic processes *dendrons*.[1]

---

[1] The latter have been known as "dendrites," but the termination "on" is for various reasons preferable to "ite."

FIG. 1.

DIAGRAM SHOWING THE PROBABLE RELATIONS OF SOME OF THE PRINCIPAL CELLS AND FIBRES OF THE CEREBRO-SPINAL SYSTEM TO ONE ANOTHER.



1, a cell of the cortex cerebri; 2, its axis-cylinder or nerve-process (neuron) passing down in the pyramidal tract, and giving off collaterals, some of which, 3, 3, end in arborisations around cells of the anterior horn of the spinal cord, the main fibre having a similar ending at 4; *call.*, a collateral passing in the corpus callosum to the cortex of the opposite side; *str.*, a collateral passing into the corpus striatum; 5, axis-cylinder process (neuron) of anterior cornu-cell, passing to form a terminal arborisation in the end-plate of a muscle-fibre, *m.*

6, a cell of one of the spinal ganglia. Its neuron bifurcates, and one branch, 7, passes to the periphery to end in an arborisation in the sensory surfaces. The other (central) branch bifurcates after entering the cord (at 8), and its divisions pass upwards and downwards (the latter for a short distance only); 9, ending of the descending branch in a terminal arborisation around a cell of the posterior horn, the neuron of which, again, ends in a similar arborisation around a cell of the anterior horn; 10, a collateral passing from the ascending division directly to envelop a cell of the anterior horn; 11, one passing to envelop a cell of Clarke's column; 12, a collateral having connections like those of 9; 13, ending of the ascending division of the posterior root-fibre around one of the cells of the posterior columns of the bulb; 14, 14, neurons of cells of the posterior horn passing to form an arborisation around the motor cells; 15, a fibre of the ascending cerebellar tract passing up to form an arborisation around a cell of the cerebellum; 16, neuron of this cell passing down the bulb and cord, and giving off collaterals to envelop the cells of the anterior horn; 17, neuron of one of the cells of the posterior column of the bulb passing to the cerebrum, and forming a terminal arborisation around one of the smaller cerebral cells; 18, neuron of this cell, forming an arborisation around the pyramidal-cell, 1. All the cells here represented, except 6, also possess dendrons, but these are mostly represented as cut short near the body of the cell, so as to avoid complicating the diagram.

And combined processes, such as occur in the motor nerve-cells of arthropods (31) can conveniently be known as *neurodendrons*. It must clearly be understood however that all processes of nerve-cells are ultimately dendritic. Almost without exception the neuron or nerve-fibre process, although it may have a course of several feet without giving off a branch, finally ends in a terminal arborisation (fig. 1). Examples of this fact occur readily: *e.g.*, the arborisation of the nerve-ending in muscles: the arborisation of nerve-fibrils in sensory structures, such as the cornea of the eye, and the epidermis of the skin. Even when a nerve-fibre apparently ends in a simple extremity it can generally be noticed, as in the corpuscles of Pacini, and in the tactile corpuscles of Meissner, that the actual ending of the essential part of the nerve-fibre is really arborescent.

It has been usual to classify nerve-cells according to the number of processes which pass off from the cell-body into uni-polar, bi-polar and multi-polar.[1] This classification is not,

[1] Cajal (30) has recently spoken of "apolar" nerve-cells, but this is completely a misnomer, for the cells in question (spongioblasts of the retina) have a well-marked process, which ramifies dendritically. What Cajal intends to imply is that they are devoid of an axis-cylinder process, but it appears to me that the nervous nature of these cells is very doubtful.

however, a natural one and does not distinguish between the kinds of processes given off from the cell-body, which ought to be done in any system of classification. Thus there are some nerve-cells which have no dendrons; there are others which have many dendrons. All, however, possess at least one neuron. We may then primarily distinguish between *dendric* and *adendric* cells; and according to the number of neurons we may further distinguish these cells into those which are *mononeuric* and those which are *polyneuric* (*dineuric*, *trineuric*, etc.), according as we find one, two, or more neurons or axis-cylinder processes passing off from the cell-body or from any of the dendrons. The majority of cells which have been hitherto observed belong to the mononeuric class. As examples of this we may take the cells of the anterior horn of the spinal cord, many of the cells of the cerebral cortex, and the large cells of Purkinje of the cerebellum. But on the other hand, we have examples of cells with more than one axis-cylinder process (fig. 2) in some of the superficial cells of the cerebral cortex (29), and obviously also in the well-known "bi-polar" cells of the spinal ganglia of certain fish, and of the embryos of vertebrata generally, in the nerve-cells of the neuro-muscular sheet of Aurelia (43), and in other cells which I will not stop now to enumerate.

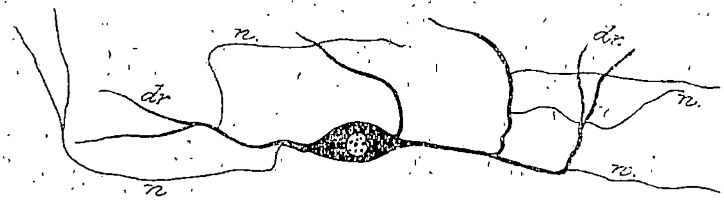


FIG. 2.

A NERVE-CELL FROM THE SUPERFICIAL OR MOLECULAR LAYER OF THE CEREBRAL CORTEX (after R. y Cajal.)

n, *n*, neurons; *dr*, *dr*, dendrons.

Starting, then, with the two kinds of nerve-cell, dendric and adendric, we can classify them further into those which are dendric and mononeuric, such as the cells of the anterior horn of the spinal cord, those which are dendric and polyneuric, such as some of the superficial cells of the cerebral cortex already mentioned; those which are adendric and mononeuric, as in the spinal ganglia of man, and those which are adendric and polyneuric such as the dineuric or bipolar cells above mentioned. The cells of sympathetic ganglia have hitherto usually been believed to be adendric and polyneuric, but we must now, as it would appear from recent observations of Cajal (30), Retzius (34), and Gehuchten (7), consider them as in reality dendric and mononeuric (fig. 3). In this way we obtain a classification of nerve-cells, firstly according to the kinds of processes which they possess, and secondly according to the number of neurons: the number of dendrons appears to be of altogether subordinate importance.

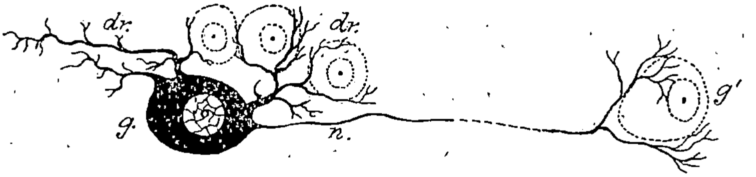


FIG. 3.

DIAGRAM OF A SYMPATHETIC GANGLION CELL (after G. Retzius.)

g., body of the cell; *dr.*, its dendrons; *n.*, its neuron, ending in a ramification which invests another cell (*g'*).

Nerve-cells may be further distinguished into those which possess relatively short neurons, and those which possess relatively long neurons. This distinction gives rise to the two types of nerve-cell which were first described by Golgi (9, 10, 11), and which were by him regarded as motor and sensory respectively. As is now well known, Golgi regarded the cells which possess long axis-cylinder processes (*e.g.*, the cells of the anterior horn of the spinal cord, and the projection-cells of the grey matter of the cerebral cortex) as being concerned mainly in the giving out of efferent impressions; whereas he regarded the cells (usually smaller in size) possessing relatively short and soon-branching nerve-fibre processes, as essentially sensory or receptive in function. But there can be little doubt that this distinction into motor and sensory cells which was drawn by Golgi, can no longer be accepted. At the same time there may still be a functional difference between the two kinds of cell, but this functional difference may perhaps better be expressed by using the term *projection-cell* for the cell of Golgi's first type with long axis-cylinder process, and the term *intermediary cell* for the cell belonging to Golgi's second type, with relatively short axis-cylinder process. By the latter term is implied that the cell in question offers an intermediary link between centripetal impressions which may be brought to a nerve-centre by the neuron of a sensory projection-cell, and the centrifugal impressions which pass away from the nerve-centre by the neurons of motor projection-cells.

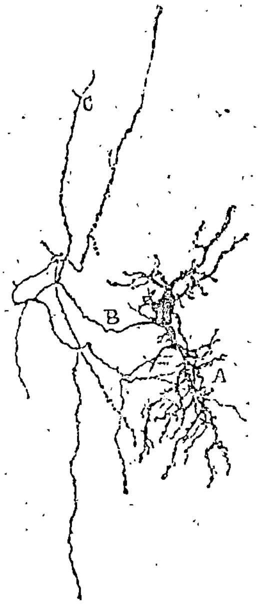


FIG. 4.

AN INTERMEDIARY NERVE-CELL FROM THE POSTERIOR HORN OF THE SPINAL CORD. (R. y Cajal).

A, dendrons; B, neuron, soon ramifying, and its branches ending either simply or in bifurcations (as at *c*).

With respect to the structure of the cell-processes, it is impossible to say positively that there is any essential difference between the neurons and dendrons, but there are certain minor differences which can be noted. One of these is the mode in which lateral processes are given off from main trunks. From the dendrons these usually come off at triangular enlargements, from the neurons without such enlargements. The dendrons usually begin to branch quite close to the cell-body, but the neuron also, which was supposed at one time not to branch until it reaches its termination, often gives off fine lateral ramuscles very early in its course. To such fine branches of the neuron, whereever occurring, the name of *collaterals* has been given by Cajal; this is a convenient term and one which may very well be adopted.. There is further often some difference in appearance between the two kinds of process when prepared by the Golgi method of staining, so that an experienced observer can detect in the midst of an interlacement of fine fibres derived from the ramifications of the dendrons of nerve-cells, neurons of the same or of other cells, traversing the grey matter through the interlacement; but it is not easy always to say what it is which confers this difference of appearance. It is, perhaps, due to the fact that the neurons remain almost unaltered in diameter along their course, whereas the dendrons, from the manner in which they branch, become gradually smaller as they are traced inwards. Moreover, the dendrons have usually a rough outline, while the neurons are more commonly smooth. Some of these differences are very manifest in the cells of the cerebral cortex (fig. 5).

Both neurons and dendrons have an apparently fibrillar structure. This structure has been mainly investigated in the neurons as they occur as axis-cylinders of the medullated nerve-fibres. The current idea of the structure of the axis-cylinder of the nerve-fibre, is that it is composed of a bundle of fine fibrils, which form the true conducting part of the nerve-fibre, and which are embedded in a clear substance comparable to the clear substance of protoplasm. These fibrils—ultimate fibrillæ of Max Schultze—have usually been regarded as completely distinct from one another; but the fact must not be ignored, that in the opinion of many

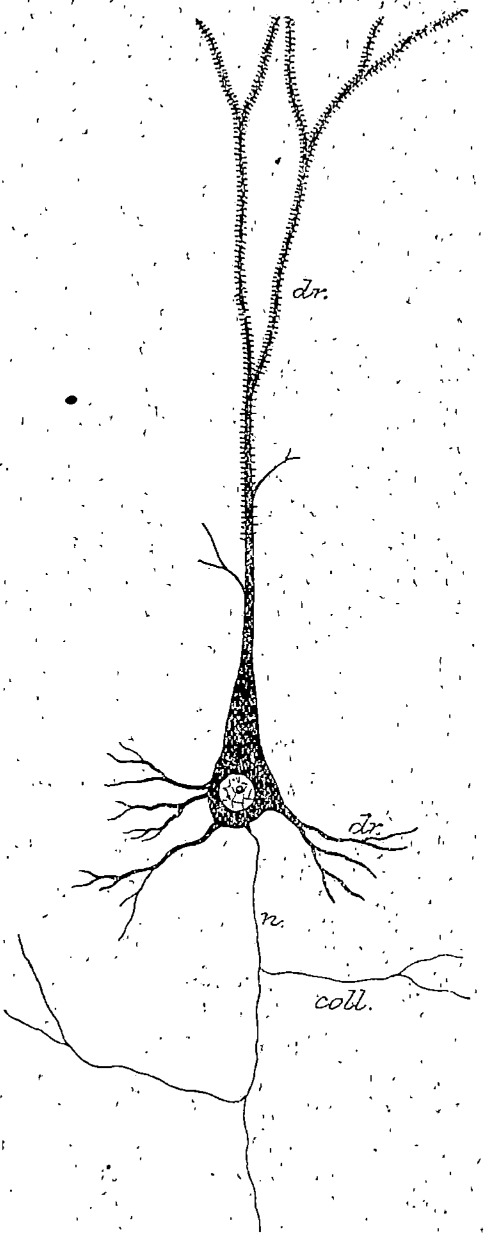


FIG. 5.

A PROJECTION-CELL OF THE CEREBRAL CORTEX.
dr, dendrons; *n*, neuron; *coll.* collaterals.

histologists (Heitzmann, Leydig, Frommann, Nansen) the nerve-fibrillæ of the axis-cylinder are nothing but a repetition of the reticulum of fibrils which is described as occurring within the protoplasm of all cells; the reticulum having, in this case, been drawn out to such an extent that the meshes of the reticulum become extremely elongated and the fibrils of the reticulum, to all appearance, parallel and distinct. In fact, according to Nansen (25), the apparent fibrils are really the optical longitudinal sections of sheaths or septa of spongioplasm, which subdivide the fibre into tubes filled with hyaloplasm, which forms the true conducting material of the nerve-fibre. The observations of Max Schultze, however, upon the structure of the non-medullated fibres of the olfactory nerve and of the axis-cylinder of the ordinary medullated fibres (fig. 6), indicate, with considerable clear-

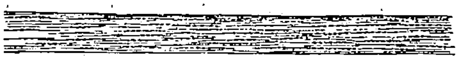


Fig. 6.

Varicose nerve fibrils seen within an axis-cylinder. (M. Schultze).

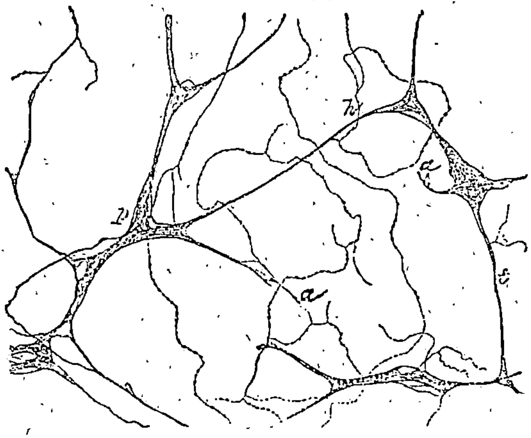


Fig. 7.

Varicose nerve-fibrils as seen in a chloride of gold preparation of the cornea. (Ranvier).

ness, an anatomical discontinuity of the individual fibrils, and this is further borne out when we trace the ramification of the nerve-fibre at its peripheral extremity. Here, in many instances, as for example in the cornea (fig. 7), and even in the nerve-endings of the motor-nerves upon the so-called end-plates, we can observe a complete separation of the fibrils which have composed the axis-cylinder and which are here ending in the ultimate branchings of the axis-cylinder.

I have here a representation of a photograph from a section of nerve-fibres in which the fibrils appear distinct from one another, and tubular in structure. In the photograph, which was taken with an apochromatic oil-immersion objective of Zeiss, the section of each fibril shows a minute circular clear area bounded by a definite line. I know no other way of interpreting this appearance than by assuming it to be really a minute tubule. And it seems to me also that the appearance of the terminating fibrils, as we see them in nerve-arborisations which have been treated by various re-agents, and as they have been represented by Max Schultze in broken axis-cylinders after treatment with osmic acid, bears out this conclusion, for it is difficult otherwise to understand why we should invariably find upon such nerve-fibrils the varicosities which every histologist knows to be so characteristic of them. It is easy on the other hand to comprehend the production of such varicosities if we imagine the fibril to be an excessively fine-walled tubule

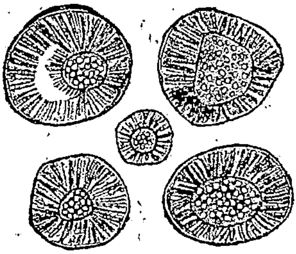


FIG. 8.

SECTION OF NERVE-FIBRES SHOWING THE TUBULAR APPEARANCE OF THE FIBRILS OF THE AXIS-CYLINDERS.

filled with fluid; in fact it would be almost impossible to suppose that under the influence of re-agents such minute and thin-walled tubules should not take on a varicose form. This is a question which we cannot at the moment decide; but it is one of some interest with regard to the possible manner in which nerve-impulses are conducted along nerve-fibres.

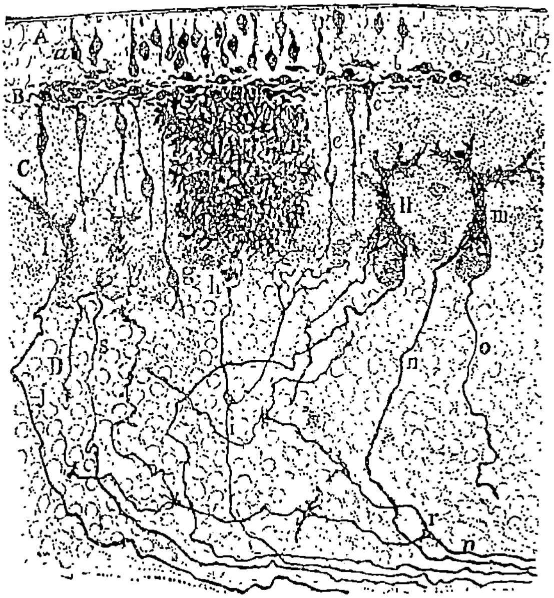


FIG. 9.

TRANSVERSE SECTION OF THE CEREBELLUM OF A 16-DAY OLD PUPPY, GOLGI'S METHOD. (Ramón y Cajal).

A, epithelium-like layer of superficial zone; B, layer of horizontal bipolar cells; C, molecular layer; D, granule layer.

a, epithelium-like cell; *b*, bipolar horizontal cell; *c*, cell sending a process downwards into the molecular layer; *e*, *f*, *g*, bipolar vertical cells; *h*, cell of Purkinje; *i*, its nerve-fibre process giving off a collateral towards the molecular layer; *j*, *n*, *o*, *s*, fibres from the white substance passing to form plexuses which envelop the upper part of the bodies and the proximal part of the dendrons of cells of Purkinje; *r*, "moss" fibre; *p*, *q*, moss-like expansions on its course and at its extremity.

The next part of our subject to come under consideration is the relation which nerve-cells bear to one another. It was formerly supposed that all nerve-cells were united with one another by distinct processes, and when it was shown by the careful preparations of Gerlach and other observers, that this direct union of one nerve-cell with another by relatively coarse fibres or processes does not obtain, it was still believed that they became united by means of a fine network which was supposed to pervade the whole grey matter of the nervous system, and which was believed to be formed by the ramifications of the dendrons uniting with one another. We can now, however, state by virtue of observations which have accumulated since the employment of Golgi's method of staining, that firstly, there is never a direct union of nerve-cells by comparatively coarse fibres;[1] secondly, there is not even a union of nerve-cells by means of a net-work of fine fibrils; and thirdly, that every nerve-cell with all its processes is a distinct and isolated anatomical unit.[2] We can further state with great probability that the only connection of one nerve-cell with another

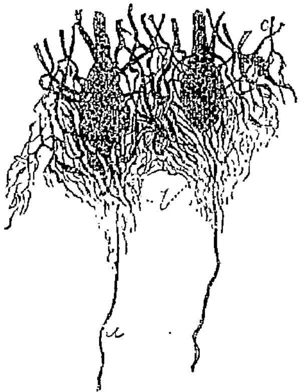


FIG. 10.

BASKET-WORK OF FIBRES AROUND TWO CELLS OF PURKINJE. (Ramon y Cajal.)

a, axis-cylinder or nerve-fibre process of one of the corpuscles of Purkinje; *b*, fibres prolonged over the beginning of the axis-cylinder process; *c*, branches of the nerve-fibre processes of cells of the molecular layer, felted together around the bodies of the corpusles of Punkinje.

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[1] The case of twin-cells, such as are found in the rod and cone layer of some animals, and such as Fritsch (5) has described in the spinal cord of the electric fish, Mormyrus, are only apparent exceptions to this rule.

[2] It was to this distinct isolated anatomical unit that Waldeyer proposed to devote the term "neuron."

is a physiological one, and that it takes place by the adjunction of the arborised process or processes of one nerve-cell, either with the cell-body of another cell, as in the cerebellar cortex (figs. 9, 10) and in various other parts, or by the adjunction and interlacement of the arborised processes of one nerve-cell with similar arborised processes of other cells, as in the olfactory glomeruli (fig. 11, *gl.*) In fact we may regard the basis of the grey matter of the nervous system—the granular-looking substance in which the nerve-cells are embedded—as an extremely fine interlacement of ramified processes, not only of the nerve-cells which actually lie in that particular grey matter, but also of nerve-cells which lie in other parts of the nerve-centres or even in the peripheral parts of the nervous system, and which on arriving at the grey matter similarly break up into a fine arborescence of nerve-fibrils.

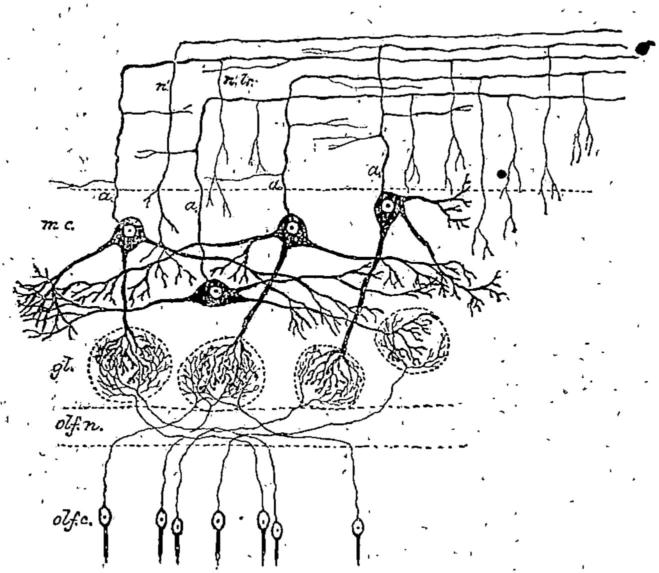

FIG. 11.

DIAGRAM OF THE CONNECTIONS BETWEEN THE OLFACTORY SENSE ORGAN AND THE BRAIN.

olf.c., olfactory cells sending their neurons *olf.n.* into the olfactory glomeruli, *gl*; *m.c.*, "mitral" cells of olfactory bulb, sending dendrons into the glomeruli, and neurons, *a*, *a*, into the olfactory tract, *n. tr.*; *n.'*, a neuron derived from a cerebral cell, ending in the olfactory bulb.

This view is extremely probable by reason of the numerous observations which have recently accumulated on the disposition of nerve-fibres and nerve-cells within the grey matter of the vertebrate nerve-centres (6, 17, 20, 27), and it is rendered still more probable by what we now know regarding the granular-looking substance which forms a large portion of the ganglia of the central nervous system of invertebrates and which was termed by Leydig, who first carefully described, it the *Punktsubstanz*. Within this "Punktsubstanz" (dotted substance) it has long been known that many of the processes of the nerve-cells terminate, and it is now recognised by the employment of new methods, and particularly the method of Ehrlich, and the method of Golgi, that the

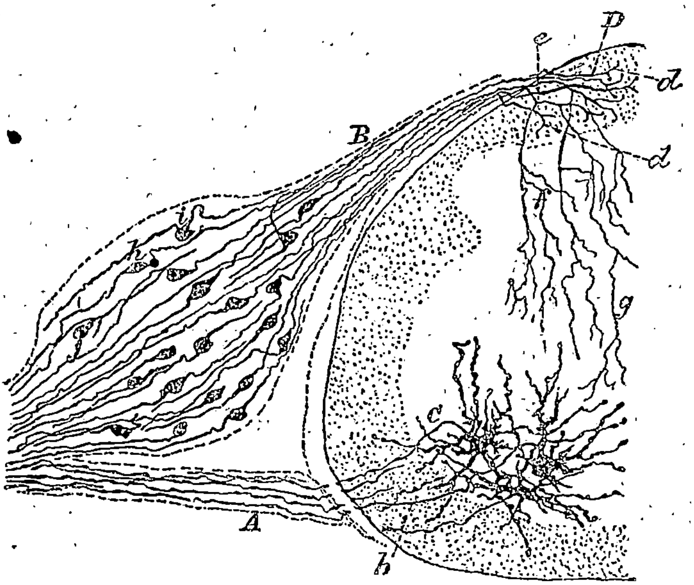

FIG. 12.

TRANSVERSE SECTION OF THE SPINAL CORD OF A CHICK ON THE 9TH DAY OF INCUBATION, PREPARED BY GOLGI'S METHOD. (Ramón y Cajal).

A, axis-cylinders of anterior root-fibres issuing from large cells of the anterior horn, *C*.

B, Posterior root-fibres passing from the bi-polar cells of the spinal ganglion, into the posterior column of the spinal cord, *D*, where they bifurcate, *d*, and become longitudinal; *e*, *f*, *g*, collaterals from these fibres, passing into the grey matter.

Punktsubstanz is entirely made up of the finely ramified and somewhat varicose terminations, not only of the processes of the nerve-cells of the ganglia which constitute the central nervous system in these animals, but also of processes which arrive at those ganglia from nerve-cells at the periphery. I would refer you, in substantiation of this fact, to the accompanying figure (fig. 13), after Retzius (31); showing the dotted substance in one of the ganglia of Astacus. In this it will be seen that the so-called Punktsubstanz is mainly made up of ramifications of fibres derived from the neuro-dendrons of the large motor nerve-cells. Similar observations have been made by Rohde (40) and by Biedermann (2).

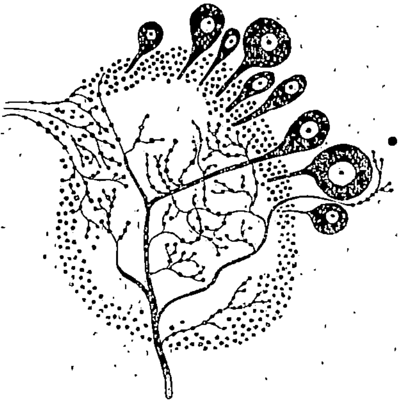

FIG. 13.

DOTTED SUBSTANCE OF LEYDIG IN ASTACUS. (After a figure by G. Retzius).

We may next consider the *functions* of the nerve-cell, and in doing this it will be convenient provisionally to consider the functions of the cell-body and cell-processes distinctly.

The first and most prominent function of the body of the nerve-cell is unquestionably that of nutrition; and this function is in all probability dependent upon the presence of the nucleus. Many instances could be given from cells other than nerve-cells of the dependence of the nutrition of cell-protoplasm on the nucleus. Thus it has been shown by Hofer that when an amœba is separated into two parts, one of which contains the nucleus and the other is deprived of a nucleus, the part which contains the nucleus will continue to live, to take in nourishment, to grow; will in fact continue to perform all the functions of the cell; whereas the part which is deprived of the nucleus, although it does not immediately die, and although it may still exhibit in a less active manner many of the phenomena of amœboid movement, yet fails to take in nourishment, fails to form more protoplasm or another nucleus, and although it may continue for a time to exist, ultimately perishes, probably from inanition. It has also been shown that vegetable cells (or portions of such cells) which are deprived of a nucleus, although they also can continue to live for a time, no longer carry on their nutritive functions as before, and that such portions of cells, although they may comprise the greater part of the original cell, have entirely lost the power of forming a cellulose wall, a function which was possessed by the original cell, and is still maintained by the portion of cell which yet contains a nucleus. And it has lately been shown by Hodge (15), who subjected the fibres of the posterior roots of the spinal nerves to prolonged stimulation, that this continued activity of the fibres is accompanied by marked wasting of the nuclei of the cells of the spinal ganglion, as well as by certain alterations in the cell-protoplasm; changes which are probably produced by the increased activity of their trophic functions, for as we shall presently see, the nerve-impulses do not, in all probability, actually traverse the cell-bodies of these cells. It is not therefore surprising to find that when a portion of a nerve-cell, *e.g.*, one of its processes, is separated from the cell-body containing the nucleus, that portion of the nerve-cell, although it may continue for a time to live, and even to conduct nerve-impulses when these are artificially produced in it, ultimately dies. This is in fact the basis of what is known as the Wallerian degeneration of nerve fibres. When we completely recognise that a nerve-fibre is nothing but the process of a nerve-cell, we understand immediately how it is that when a nerve-fibre is severed the part which is cut off from connection with the parent cell must ultimately die. This is found to be an invariable rule and it matters not whether the part of the fibre which is thus severed from the cell conducts impulses towards or from a nerve-centre; in both cases the result is the same—the process of the cell dies. It involves in its death, or at least in the changes which accompany its death, certain changes in the sheaths of the nerve-fibres which enable us with great readiness to recognise such degenerating fibres; and this the more easily from the method which Marchi has introduced, and by means of which we can stain deeply the myeline sheaths of such nerve-fibres in the earlier stages of their degeneration, whilst leaving unstained the myeline sheaths of normal fibres. This nutritive function of the body of the nerve-cell has not always been so completely recognised as it deserves, but has been of late strongly insisted upon by Nansen (25), who has even gone so far as to deny to the cell-body any other function than that of presiding over the nutriment of the whole cell and especially over that of the axis-cylinder process. It is clear, however, that although the function of nutrition may be the one essential function of the nucleated body of the nerve-cell, many cases occur in which the body of the cell also serves for the transmission of nerve-impulses. That must be so with the so-called bi-polar cells, where the cell-body gives origin to two nerve-fibres passing in opposite directions; the impulses which course along these nerve-fibres necessarily traverse the body of the cell. And this must also be the case in the motor projection-cells of the spinal cord and bulb where each nerve-cell gives off only one unbranched neuron. In this instance nerve-impulses are probably communicated to the body of the cell from the interlacement of nerve-fibres, derived from other cells, which enwrap the body of the motor cell. Granting, however, the possibility of the transmission of nerve-impulses through the cell-body it is quite open for us to believe that this is, if one may say so, more or less accidental. It is certain that there are many instances in which nerve-impulses can be conceived to be transmitted along nerve-fibres without in the course of that transmission passing through a nerve-cell at all. This is in all probability the case with the cells of the spinal ganglia of higher vertebrates. From these cells, as is well known, only one process passes off and this process, as was first shown by Ranvier, bifurcates, one branch passing towards the periphery and the other towards the nerve-centre. Sensory impulses which originate in the periphery and pass along the fibre can perfectly be conceived as being conducted directly to the nerve-centre, past the T-shaped junction with the process of the nerve-cell, and without passing through the nerve-cell at all. It has indeed been actually shown by Langley and Anderson (18) that the passage of nerve-impulses along the fibres of the posterior roots is not blocked by a dose of nicotine, which would be more than sufficient to block their passage through sympathetic ganglia. And in the case of invertebrates, such as the earth-worm, the structure of whose nervous system has recently been elucidated by Lenhossék (19) and Retzius (32), it is clear that sensory impulses which are brought by the fibres from the integument to the central nervous system may perfectly well become converted into motor impulses within the Punktsubstanz without necessarily traversing the motor nerve-cells at all; the latter being only connected with the Punktsubstanz by dendritic collaterals, which pass off from their large neuro-dendric processes (fig. 14).

It is, however, almost universally held that nerve-cells may act as generators of nerve-impulses: that in fact they may function as independent nerve-centres. But it is by no means easy to bring forward direct proof of this action. Perhaps the so-called automatic action of nerve-centres would be regarded as the best instance of such intracellular genesis of nerve-impulses, and, as an example of automatic action, the action of the respiratory centre in the medulla oblongata (which continues to convey nerve-impulses along the efferent channels when all afferent channels to the cells have been cut) may be taken. But this action of the respiratory centre is probably due to the chemical stimulation which the centre receives from the venosity of the blood circulating in the vessels of the medulla oblongata. The stimulus is supposed to affect the nerve-cells which give origin to the fibres passing towards the respiratory muscles, or at least to those spinal segments from which the fibres to the respiratory muscles arise: it practically amounts to an external stimulus applied to the nerve-cells and is comparable to the application of an artificial stimulus to the same part of the nervous system, such, for example, as the chemical stimulus of salt, or the electrical stimulus derived from an induction-coil. The action of the nerve-

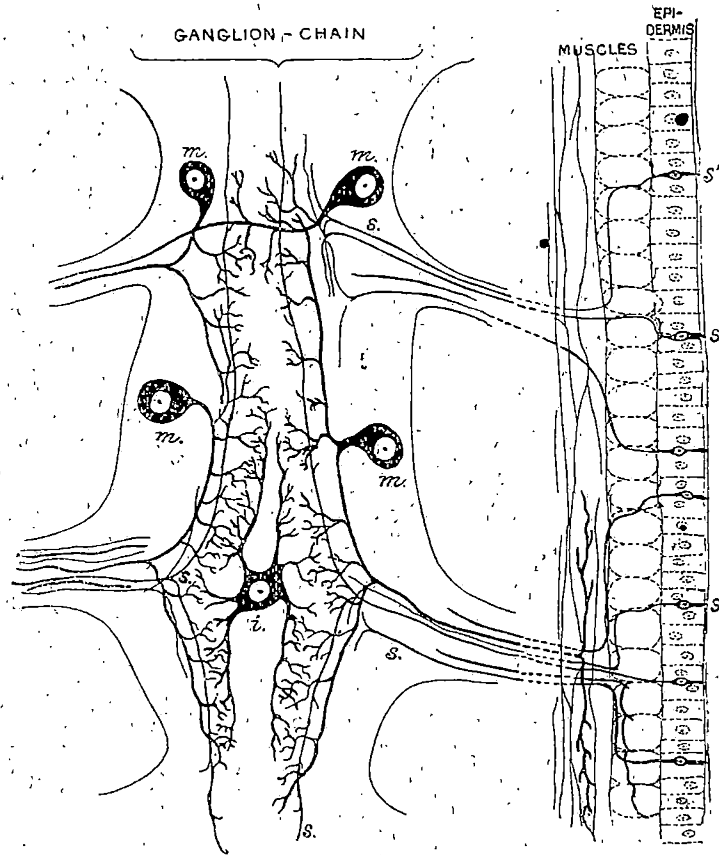

Fig. 14.

Diagram of the nervous system of Lumbricus. (After Lenhossék and Retzius).

m, motor nerve-cell; *i*, intermediary cell; *s*, sensory fibres proceeding from *s'*, sensory cells in the epidermis.

cells cannot therefore be said to be truly automatic: in other words, the nervous impulses do not arise *de novo* without any obvious external cause by the action of the cell-substance. And I think that it would be difficult to instance any "automatic" action which might not also be referred either to a chemical stimulus due to variations in the blood or to stimuli reaching the centres from the periphery, or from some other parts of the nerve-centres.

There is no doubt, however, that on due stimulation, whether mechanical, chemical, or electrical, the body of a nerve-cell may produce nerve-impulses. This, of course, is only to be expected, since the processes of the cells are capable of producing nerve-impulses under similar conditions. It is a function which is not special either to the cell-body, or to the processes, but is common to both. We may, however, fairly consider whether there is no difference in the performance of this function in the cell-body as compared with its performance by cell-processes.

Unfortunately, our knowledge of nerve-impulses is extremely limited. We know their rate of transmission; that they are transmitted in a wave-like manner, and can pass in either direction along nerve-fibres; and, further, it is probable, although not absolutely proved, that the passage of nerve-impulses is accompanied by chemical processes. With regard to nerve-cells we have evidence of a more or less positive character on this head. It has been shown that the grey matter of the nerve-centres becomes acid after it has been excited by drugs or by other methods, and that it also, when it has been called into activity, produces a notable rise of temperature. This rise of temperature was shown to the Royal Society last year, by Mosso (24). At the same time, the opinion has been largely held that nerve-fibres themselves do not participate in these chemical processes of the nerve-cell. Points in favour of this view are chiefly derived from the fact that various observers, who have investigated the conditions of nerves during the passage of nervous impulses, have come to the conclusion that nerve-fibres cannot be fatigued.[1] And it is now believed that fatigue, both in muscles and in the nerve-centres, is produced by the accumulation of the products of chemical processes; if, therefore, nerve-fibres show, even after prolonged activity during many hours, no sign of fatigue, it is probable that the chemical processes which produce fatigue within the nerve-centres, do not occur within the nerve-fibres. At the same time the latest observations upon this subject, those of Edes (3), have shown that after some hours of excitation the action-currents which result from the excitation tend to become weakened, and since these action-currents may be taken as a sign of nerve-activity, this would be an indication that even in peripheral nerves prolonged excitation may ultimately produce fatigue. Rolleston (42) endeavoured to obtain evidence regarding the same question in a different way. Determining by a delicate method whether the passage of nerve-impulses along the fibres is accompanied by a development of heat, he was unable to obtain evidence of any such warming. But it must be noted that Rolleston found that when the nerve was dying there was evidence of the formation of heat, and since the chemical processes which accompany death of living tissue appear to be very similar to the chemical processes which accompany activity, as is seen, for example, in the case of muscle, it is very possible that there may still have been thermal changes proceeding in the living nerve when excited, but that these were of too slight a nature to be detected owing perhaps to the very small amount of true conducting matter within a peripheral nerve as compared with the bulk of nerve-sheaths and connective tissue structures. Perhaps also for the same reason, as well as the fact that alkaline lymph surrounds the nerve-fibres, it is impossible to determine that a nerve which has been tetanized for some time shows any sign of acidity. But, as we have already noted, when the central nervous system is thrown into activity, the white matter, which is formed wholly of nerve-fibres, becomes acid as well as the grey cortex. Since a nerve-fibre is only a prolonged part of a nerve-cell, and since we know, firstly, that nerve-cells may undergo fatigue, secondly, that chemical processes go on both in the white and grey matter of nerve-centres; thirdly, that heat is developed in grey matter, and that this heat is due, probably, to chemical processes, it is reasonable to suppose that similar chemical processes may go on in axis-cylinders of nerve-fibres but that, for the reasons we have above indicated, it has hitherto proved impossible to detect them. Moreover, the apparent non-fatigueability of nerve-fibres may be partly due to the ready reparative processes of the axis-cylinder, which may derive material for such reparation from the substance of the myeline sheath.

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[1] The literature of this subject is referred to by Edes (3).

Before considering further the manner in which nervous impulses are produced within the bodies or the processes of nerve-cells, it may be well to enquire what is the nature of a nervous impulse. It is usually assumed that nervous impulses are either waves of molecular vibration or of chemical action (that is to say, of explosive change), or of the two combined. There is absolutely no evidence in favour of the first supposition, although, as we have just seen, there is some indication that they are accompanied by chemical action. We must not, however, ignore the possibility that they may be waves of pressure or surface-tension change. The supposition that the electrical phenomena of active muscle and nerve are produced by variations of surface-tension passing in a wave-like manner along the fibres, has, in fact, been recently insisted upon as probable by d'Arsonval (1). Such variations must necessarily occur when in an elastic tube with fluid or semi-fluid contents, pressure-waves are in any way set up. But we have already had evidence that the nerve-fibrils may be extremely fine tubules (compare p. 145), and in such tubules we can readily understand how waves of surface-tension change may be possible.

Assuming that nerve-impulses may be waves of pressure or surface-tension change, we may next conjecture in what manner they might originate. Such waves might be produced by the contraction of the nerve-cell, or of any of its processes. It is certainly true that except in the cone-cells of the retina, this contraction has not hitherto been observed; but, on the other hand, it may generally be so slight as to escape observation, or it may be confined to the fibrillæ. If such contraction were assumed to be rhythmic, we could then understand how it happens that from many nerve-cells, nerve-impulses pass along nerve-fibres with a regular rhythm. This is seen in the motor nerve-cells of the spinal cord, for it is found that a rapid stimulation of the excitatory area of the brain or of the fibres of the pyramidal tract—a stimulation which applied to a motor nerve directly is sufficient to produce complete fusion of the muscular contractions, and therefore complete tetanus—is, when applied to the brain, or to the pyramidal tract, responded to by an incomplete tetanus of the voluntary muscles—a tetanus having a fairly regular rhythm of about 10 per second.

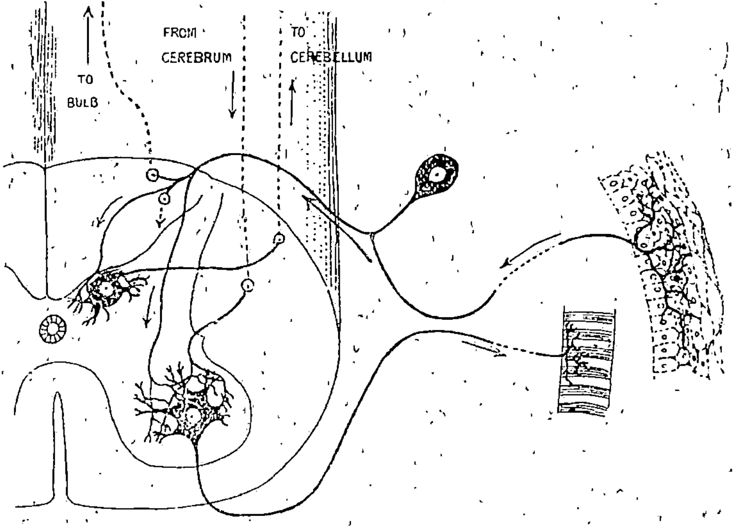

Fig. 15.

Diagram of the relations of motor and sensory cells within the spinal segment, and their connections with the higher nerve-centres.

The annexed diagram (fig. 15) shows the anatomical mechanism which probably takes part in this experiment. When a fibre of the pyramidal tract is excited, the nerve-impulses which are generated in that fibre, and which are probably of the same rate as the excitation, pass down to the grey matter of the spinal cord, and are there converted into nerve-impulses which may have a very much less frequent rhythm. This can only, so far as appears, take place at the adjunction of the terminations of the pyramidal-tract fibres with the motor nerve-cells; and it would appear that the motor nerve-cell is stimulated by the nerve-impulses which are conveyed along the fibre of the pyramidal tract, but that it responds to that action with a very much slower rhythm than that of the assumed excitation; for the excitation may be as rapid as 100 per second, or more, but provided it is not too intense the impulses which pass along the motor fibres are only at the rate, as shown by the response of the muscle, of about 10 per second. The same thing is seen when the muscles are made to contract by a reflex excitation of the skin. Such an excitation may be very rapid or it may even be continuous. This rapid or continuous excitation of the skin produces in the sensory fibre nerve-impulses which may be assumed to be at least as rapid as the excitation itself, and these are conveyed to the grey matter of the lower nerve-centres, and are converted into nerve-impulses of a relatively slow rhythm, as shown by the rhythm of the reflex muscular response. This transformation may be assumed to occur either in the motor projection-cell, or in an intermediary cell, if any such intervene between the afferent fibre and the motor-cell, and the slow rhythm of the epileptiform convulsions which follow strong electrical excitation of the cerebral cortex, and which certainly originate in the cells of the cortex, furnishes another well-marked instance of rhythmic production of nerve-impulses by nerve-cells.

There is another function of nerve-cells which we have yet to consider, and that is the reception of impressions from the exterior. All nerve-cells which receive impressions from the exterior, may provisionally be termed sensory cells although it does not necessarily follow that the impression may ultimately affect the sensorium. The sensory cells with which we are most familiar, are those of the spinal ganglia. Here (fig. 16, TACTILE) the sensory impression is not received by the body of the cell, but by a peripheral process, which passing either to a special end-organ such as a tactile corpuscle or end-bulb, or insinuating itself between the elements of the integumental and other structures, receives the impressions which cause nerve-impulses and transmits those impulses upwards towards the nerve-centres. It is not only the case with those sensations which are received through the surface of the skin or by the action of the muscles, that sensory impressions are in the first instance communicated to processes of nerve-cells; for we find from the recent observations of Retzius [33, 37] that the same is true for the auditory and for the gustatory organ (fig. 16), the nerve-terminations in which do not, as was at one time supposed, emerge from the receptive hair-cells, but really originate from bi-polar or uni-polar cells which are placed somewhere in the course of the sensory nerve, and which resemble the cells which are found upon the spinal ganglia in sending a peripheral process to penetrate between the (somewhat modified) cells of the epithelium, and a central process to penetrate the grey matter of the nerve-centres.

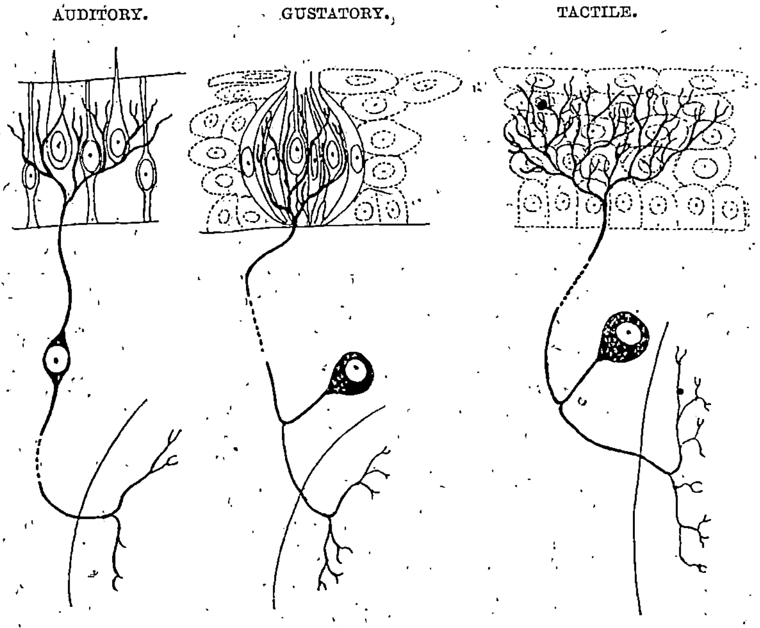

FIG. 16.

NERVE-CELLS OF SPECIAL SENSE. (Modified from G. Retzius).

In other sense-organs the nerve-cell itself more directly receives the sensory impression. This is so with the rods and cones, or rather the rod-and-cone-cells of the retina and with the olfactory cells (fig. 17). In these cases the sensory impression appears to be received, if not directly by the body of the cell, at least by a very short process which is immediately attached to the body of the cell and which can scarcely be termed a nerve-fibre process, but is rather a specialised part of the cell-body; while from the other end of the cell a fibre passes off which can only be regarded as a nerve-fibre and which ends, like other processes of nerve-cells, in an arborisation which becomes interlaced with the arborisations of other nerve-cell-processes, and thus allows the nerve-impulses which are generated within the sensory nerve-cells to be carried towards the nerve-centres. When one considers the differences met with in the several sense-

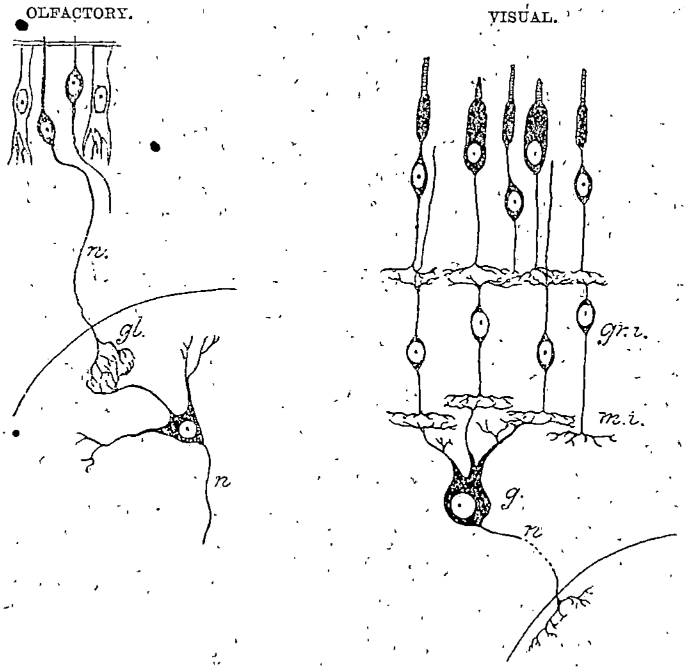

FIG. 17.

NERVE-CELLS OF SPECIAL SENSE. (Modified from G. Retzius).

n, *n*, neurons; *gl*, olfactory glomerulus; *g*, ganglion-cell of retina; *gr.i*, inner granules of retina; *m.i*, inner molecular layer of retina.

organs, viz.: that the sensations received through the skin and from the auditory and gustatory organs, are taken up by the terminal branchings of neurons, and that the sensations of light and of smell (which appear to depend upon more impalpable means of excitation), are taken up by the bodies of nerve-cells themselves, one is tempted at first to generalise from this to the effect that there is some essential difference between the two kinds of receptive organ, correlated with differences of function or of excitation. This generalisation will not, however, bear the test of further investigation, for we find from the observations of Lenhossék (19) and Retzius (32), that in the earth-worm all the sensory nerve-cells are wholly situated between the cells of the epidermis (fig. 14). There can be no doubt that these peripherally situated cells are nerve-cells, for like every other nerve-cell, each one has a neuron or nerve-fibre process (which in this case is directed immediately to the central ganglia, and ends by ramifying in the Punktsubstanz) and besides this, like many other nerve-cells, each cell has dendrons which ramify like roots underneath the epithelium but without anywhere joining the dendrons of neighbouring cells. These sensory cells are extremely numerous and are found over the whole epidermis, no other kind of nerve-termination or nerve-origin having hitherto been discovered within the integument. The nervous system of the earthworm is thus seen to be a perfectly diagrammatic structure (see fig. 14). Peripheral nerve-cells placed in the integument receive impressions and convert those impressions into nerve-impulses; the nerve-impulses are conveyed along afferent fibres to the ultimate ramification of those fibres within the Punktsubstanz, and from this Punktsubstanz they are collected either in the same form or more probably as new impulses by the ramifications of the collaterals of the neurons or neurodendrons of the motor cells and by these neurons are conveyed directly to the muscles. Now it is known that the earth-worm although it has no special sense organs, no eyes, no ears, no organs of olfaction, yet reacts to light, to sounds, to touch, and in all probability to odours. But all these impressions are operative upon the general integument and must be taken up by the sensory cells within the integument and by these converted into nervous impressions. The generalization therefore which we might be apt to assume from the study of the sense-organs of vertebrates alone is thus seen to fall to the ground. Moreover Retzius has shown (35, 36) that in other worms (Nereis) and in mollusks the sensory cells tend to lie more deeply and are no longer placed entirely within the epidermis but beneath it. They then appear like bi-polar cells, sending a (relatively short) peripheral process to penetrate between the cells of the epidermis, and a long process centrally as a nerve-fibre which enters the ganglia of the central nervous system. Such cells offer a transition between the entirely peripheral or epidermal sensory cells of the earthworm, and the bi-polar cells of the spinal ganglia of osseous fish (fig. 18).

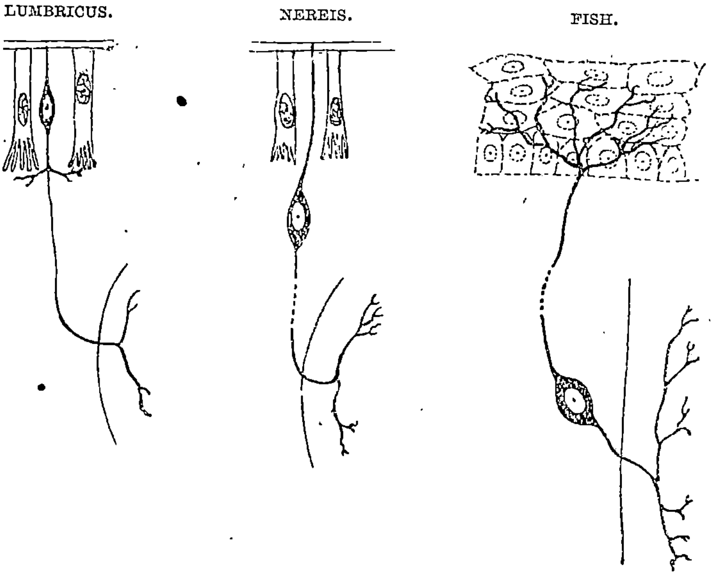

FIG. 18.

SENSORY NERVE-CELLS. (Modified from G. Retzius).

We may next consider how nervous impulses pass from nerve-cell to nerve-cell. We have seen that it is probable there is never actual continuity between nerve-cells, but only contiguity of ramified processes, either with cell-bodies or processes of other cells. Physiologically it may be shown that there is always a partial block to the passage of a nervous impulse at the conjunction of one cell with another. This is evidenced by the loss of time which occurs when nervous impulses have to pass such a block. Thus there is such lost time at the motor projection-cells of the spinal cord; for if the time taken between the excitation of fibres of the pyramidal tract, and the commencement of the contraction of corresponding muscles be measured, it will be found to be prolonged (over and above the time taken by the transmission of nerve-impulses along the given length of nerve, *plus* the time taken by inertia of the muscle) to the extent of about .01 second (or less). This lost time which occurs at the junction of the cell-chain represents the period of latent excitation of the nerve-cell. Let us consider what is probably happening at this point. The nerve-fibre to which the excitation is applied carries nerve-impulses, which become spread out in the fine arborescence, which forms the termination of that fibre and which enwraps the motor-cell. From this cell nerve-impulses start, which are not necessarily of the same rate as those which have reached the terminal arborescence just mentioned, and these new nervous impulses pass down towards the muscle and cause its contraction. It is clear that a change occurs at the adjunction of the arborescence with the cell-body. A change in rhythm certainly occurs (16), and this renders it extremely probable that the nerve-impulses which are passing off from the spinal-cord-cell are entirely new impulses. If so, we may look upon this cell as having been freshly stimulated by the impulses which have passed along the fibre of the pyramidal tract. We may briefly consider in what manner it can be thus stimulated. Since there is no evidence that the fibrils of the arborescence anywhere touch the cell-body or its processes, we must assume that a space intervenes everywhere between the two, very narrow indeed but still a space which cannot readily, if at all, be traversed by nerve-impulses. It is possible to suppose that the nerve-impulses reach the pericellular arboresence, and produce by mere induction new nervous impulses within the cell around which they play. But we have no evidence that such nerve-induction is possible. It is also open to us to suppose that the electrical change (action-current) which accompanies the passage of the nerve-impulses to the arborescence, may itself be the excitant of the nerve-cell, and that the nerve-cell may respond to this excitation by a rhythmic change of some kind, which is possibly contraction, possibly rhythmic chemical action, possibly molecular vibration, or perhaps a combination of two of these. At all events, it is probable that a new process is started within the nerve-cell. This does not necessarily follow from the fact that there is time lost at the adjunction, for a partial block to the passage of nerve-impulses and a resultant loss of time may be produced merely by mechanical means; but the change of rhythm renders it extremely probable. In considering this question, another one presents itself, viz.:—why do not nerve-impulses pass *backwards* from the motor cells of the cord to the fibres of the pyramidal tract: in other words, why are not nervous impulses generated in the terminal arboresence of the pyramidal tract-fibre, when an anterior root-fibre is stimulated? It is not a sufficient answer to this question to say one is the natural or habitual, and the other an an unnatural direction, for it has been shown by Gotch and Horsley (13)—to whom we owe our knowledge of the fact just mentioned—that nerve-impulses pass very readily from the nerve-centre *down the afferent channels*, although the strongest excitation will not cause them to pass from the efferent channels *up the spinal cord.* Since the effects pass both down and up the sensory paths, one would have expected that they would also pass both down and up the motor paths. In seeking for an explanation of this difference it has occurred to me that such an explanation may be found in the anatomical arrangement of the terminations of the pyramidal-tract-fibres around the motor cells as compared with the mode of central termination of the sensory fibres within the grey matter. It is conceivable that such structural arrangement may allow of the excitation of new nerve-impulses within the body of the motor-cell, by an electrical discharge from the fine brush of pericellular fibrils which envelopes the body of the motor-cell, whilst the electrical change which accompanies nerve-impulses up the motor fibre, when this is artificially stimulated, may be so diffused throughout the cell-body of the motor-cell, as to fail to stimulate and set up nerve-impulses in the pericellular ramification of fibrils, which represents the ending of the fibre of the pyramidal tract.

From the present consideration of this subject we arrive at the following general conclusions :

1. That every nerve-cell forms a structural element which is anatomically isolated from but in physiological continuity with other nerve-cells.

2. That the physiological continuity of these elements depends on the contiguity either of ramified cell-processes of different nerve-cells with one another, or of the ramified processes of one cell with the body of another cell. By this contiguity such isolated anatomical elements become concatenated into nerve-chains which may be either simple, as in the nervous system of the earth-worm (fig. 14), and possibly in the ordinary reflex chain of vertebrates (fig. 15), or may be complicated to any imaginable extent (fig. 1).

3. That the same nerve-impulses do not necessarily pass from one element of a nerve-chain to the next, but that more probably new impulses (often of different rhythm) are generated in the successive elements of the chain.

4. That the new impulses so generated may be set going by the electrical change which accompanies the advent of the producing impulses and are not necessarily due to the passage of those impulses themselves to the next element of the chain.

5. That either the body of the cell or any of its processes may be concerned both with the starting and with the transmission of nerve-impulses ; and, that these may originate by acts of contraction causing waves of pressure or variations of surface-tension to traverse the fibrils.

6. That the body of the cell is especially concerned with presiding over the nutrition of the whole cell-element ; this trophic function being intimately associated with the presence of the nucleus. Nevertheless nerve-impulses may both origi-

nate in and be conducted by the cell-body. The dendrons or protoplasmic processes, being extensions of the protoplasm of the cell, may primarily serve to assist in the nutritive processes, as was supposed by Golgi, but they undoubtedly also, like the cell-body itself, may in some cases convey nerve-impulses.

7. That the ordinary centrifugal paths are blocked for centripetal impulses although the centripetal paths may convey centrifugal impulses, this physiological difference being correlated with a difference of anatomical relationship at the junction of the respective cell-elements.

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ON THE VISUAL PATH AND CENTRE.[1]

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Before an audience like this, I certainly need not enlarge upon the great importance of the senses in the development of our intelligence, and indeed of our whole psychical life, of which doubtless the perceptions through our senses are the necessary conditions and basis. In order that modern psychology may possess a definite starting point and foundation, we must obtain an exact idea of the anatomical construction and physiological function of the paths and cortical areas of our senses, as well as their connections with other parts of the brain. In consequence of the law of evolution, there exists in man the highest development of the different centres, and of psychical life. For this reason we must principally study the paths and centres of the senses in *him*, whilst comparing the results obtained by experiments on animals.

For the deeper study of the senses, there is none so instructive as that of sight, which is, in certain respects, the principal one. Through the same we procure the greatest wealth of perceptions, which will continually serve as objects of our psychical activity. No sense can be so minutely examined in regard to its functions and pathological derangements, and a clinical examination will, in most cases, determine whether the lesion be of a peripheral or central nature. If we are ever to grasp the inner connection between the higher psychical activity and the construction and function of the senses, it must be principally through the deeper study of the visual path, its centre and activity. The more profound and penetrating the knowledge we obtain in this direction, the more certain will be the starting point of our psychological researches, not only in this direction, but also in that of the other senses, for we may presume that there exists a certain analogy between them. It is for this reason I take the liberty of giving a short sketch of my opinions on the above, viz. :—the visual path and centre, based upon clinical facts gathered from others as well as myself. But I shall not enter into details, and the prescribed time will not allow me to bring forward the proof for many of my views, but for further particulars I refer you to my work, "Beiträge zur Pathologie des Gehirns." The second volume has just now been published, a copy of which I now have the honour of laying before this congress.

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[1] Paper read before the Congress of Experimental Physiology in London, 1892.

In order to understand the nature of the cerebral representation of vision and its psychology, the localisation of the cortical centre for sight, is of more interest than that of the course of the visual path, which is principally an anatomical question. But the former proposition cannot be solved without a knowledge of the latter, and I shall, therefore, first discuss the question of the visual path. Moreover, for a proper comprehension of the relation of the visual centre with those of other senses, it is necessary to have an acquaintance of the connection of the visual path with the central ganglia of the brain. I will, then, in considering these anatomical questions, express myself briefly, and mainly dwell upon some of the most important points, in which I differ from current doctrines.

Enquirers have tried in different ways to arrive at a solution of the question of the visual centre. Wilbrand, for example, has by using the purely clinico-ophthalmological method elaborated a theory, which may not be unknown to my auditors. Objections might be raised against his conclusions, in that the starting point of his statements is uncertain, in this respect, that only a few of his ophthalmological cases were examined after death. Nevertheless, I have, by using the combined clinico-anatomical method, arrived at much the same conclusions. Only by this latter method, the clinico-anatomical, can we secure a firm basis for the investigation of this field of study. Time after time, it has been adopted by celebrated observers, such as Ferrier, Luciani, Tamburini, Wilbrand, Starr, Seguin, Nothnagel, Seppilli, &c., but the results at which they arrived have only partly coincided. Time will not suffice to give an historical sketch of their different opinions. I will only intimate that whilst some Frenchmen wish to extend the visual centre as far as the central convolutions, others, especially Englishmen, limit it to the occipito-angular region, and others, as Seguin and German authorities, confine it to the occipital lobe. Reinhardt would place the centre, in agreement with Munk's experiments, on the lateral surface of the occipital lobe, Nothnagel in the first occipital gyrus, Seguin on the mesial surface, and Hun places it immediately in the neighbourhood of the calcarine fissure. A new and rigorous analysis of the clinico-anatomical cases is of course necessary, and I have endeavoured to submit to it all accessible facts concerning the visual centre, and I am convinced that all these tend in the direction I propose indicating.

Besides these clinico-anatomical facts, there exists a number of other pathological ones of importance in the solution of the above problem. After extirpation of the bulbs, or a lesion of the frontal visual path, or of the external geniculate body, there arise centripetal degenerations; and after lesions of the visual cortex, arise degenerations of a centrifugal nature, which enlarge our knowledge of the centre itself. These changes have been principally studied by Monakow and myself.

Numerous investigators have devoted much time and patience in experiments upon animals, but I omit stating their conclusions here, in the belief that these renowned experiments are known by all present. Meanwhile, one should, with great circumspection only, be permitted to apply the results of experiments upon animals directly to man, especially as experimentors have not arrived at the same results.

In the first place, in order to discover the visual path and centre, every clinical case supplemented with a *post-mortem* is serviceable, in which some defect of central origin is observable in the field of vision. These defects are almost always hemianopic. But nearly as important as the positive cases are the negative, with an absence of any defect in the field of vision, but with a lesion in the neighbourhood of the visual path or centre. These latter control the conclusions drawn from the positive cases. A necessary condition, to obtain a sure result, is that the clinical and pathological examinations are conducted by competent persons, because hemianopsia is often latent and frequently impossible to detect in feeble and unintelligent patients. Since I commenced to make a systematic examination of my patients in this respect, I have found in my hospital in Upsala, nearly 40 hemianopic cases, supplemented in the *post-mortem* records; whilst Seguin, in 1886, could only discover about the same number in the whole of literature. The number of cases has considerably increased of late years, and there are now perhaps more than 160 on record. Lesions of the visual path, especially cortical ones, are also evidently very common, and we may hope that further clinical researches upon this matter will thoroughly elucidate the organisation of this sense. This, however, can only be the case, if clinical and pathological scientists work methodically. An analysis of the already published cases proves that the present manner of working does not satisfy the requirements of science, for many even of the most important cases are incomplete. My statements are chiefly based upon my own cases.

The Visual Path can be divided into the frontal, middle and occipital. I shall not dwell upon the anatomical construction of the frontal visual path, as it is only of minor importance in elucidating the problem before us. Nature has herself indicated its direction. In the optic nerves, chiasma and tracts, the fibre from different quadrants lie in separate bundles, and those from corresponding points are not immediately beside each other. Fibres from the upper retinal quadrant lie dorsally in the tracts.

The Middle Path.—The course of the optic fibres and their connection with the central ganglia has not yet been thoroughly investigated. It is very important to notice that it is necessary to distinguish between visual and optic fibres; a lesion of the former produces a defect in the visual field alone, whilst the latter are to be considered as reflex fibres. There is no doubt that the three great central ganglia, the external geniculate body, the pulvinar and the corpora quadrigemina anterior, all receive fibres from the optic tracts; but the question is, do they all receive visual fibres? Thereupon depend their several functions. The above three main ganglia have evidently different duties to perform in different animals, but I only here intend referring to their importance in man. The solution of this problem should be effected by an analysis of the respective clinico-pathological cases. The central ganglia are often injured by lesions, which are succeeded by hemianopsia, but these are often microscopical, and extend simultaneously with larger lesions into the occipital or parietal lobe, and, of course, are then overlooked.

Literature specifies at least eight[1] cases of tumour in the central ganglia, six of hæmorrhage, and sixteen of softening, accompanied with hemianopsia. An analysis of these cases gives the following results. In the case of tumour the growth was so diffuse that no enlightenment could be obtained concerning the functions of the above three main ganglia. Of the cases of hæmorrhage there were only two, Dreschfield's and my own, in which the lesion was so circumscribed that a conclusion could be drawn of the function of the pulvinar, but from the freshness of the hæmorrhage and its pressure upon neighbouring ganglia the result could but be considered uncertain. Neither are the sixteen cases of softening of use in the solution of the problem, and this for reasons you will find discussed in my book. Thus the analysis of the clinical cases with hemianopsia does not decide this question, and here, therefore, the importance of the negative cases arises. Nothnagel has, by an analysis of lesions in the corpora quadrigemina, proved that such need not give rise to any form of visual disturbance, and further, some cases go to prove that the destruction of the pulvinar is not always accompanied with hemianopsia. By means of exclusion we shall also prove that there is no clinical proof that in man the pulvinar or corpora quadrigemina ought to contain visual fibres and thus be necessary to sight. Consequently, the external geniculate body is the main sight ganglia in man. Its destruction will always produce some form of hemianopsia.

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[1] See my work "Pathologie des Gehirns," Upsala, 1892, p. 266.

The position of the fibres in this ganglia has only partially been ascertained. My own researches in a case of atrophy of one optic nerve have persuaded me that fibres from both eyes lie immediately together in this ganglia, and not in separate bundles, as in the frontal portion of the visual path. And, finally, Monakow has on account of his own enquiries expressed the opinion that the visual fibres do not lie in direct connection with the ganglia cells, but ramify in the ground substance. To the above I will only add that the clinical analysis does not render any support to the commonly accepted theory that a lesion in the posterior portion of the internal capsule gives rise to hemianopsia, and still less to the idea that a decussation in the middle line of the corpora quadrigemina takes place.

The Occipital Visual Path.—The determination of the situation and limits of the occipital visual path is of the greatest moment in deciding the debated question as to whether the parietal, and especially the angular gyrus, belongs to the sight area or not. It is very common for lesions in the parieto-angular region to be accompanied with hemianopsia, and this fact has given rise to the opinion that it belongs to the sight area. But a critical analysis of the cases tends towards another view, for in a considerable number of cases lesions in that region are not followed by hemianopsia. How, then, can we find a clue to this apparent contradiction? The explanation is as follows:—If the lesion implicates the visual path in the optic radiation of Gratiolet, it will induce hemianopsia, but not otherwise.

Where does the Visual Path lie?—This question will be solved by a clinico-pathological analysis. Literature mentions at least 13 cases of abscess or tumour in the parietal lobe. In eight of these hemianopsia was present; in five not. There are also four traumatic cases. In most of these the lesions (tumours or traumata) were so large and diffuse, or they pressed upon the optic radiation to such an extent, that their influence cannot be accurately determined. The negative cases are, again, of decisive importance, for they prove that a lesion of the parieto-angular cortex need not induce a defect in the field of vision. It is just the same in cases of hæmorrhage. Six were followed by hemianopsia, but in one of mine—that of a professor of medicine—the hæmorrhage was not accompanied with hemianopsia. Finally, there exist at least 25 cases of softening in the parietal lobe, in 12 of which hemianopsia was present; in seven absent. An analysis of all these cases gives us this result: the visual path is situated in the ventral portion of the optic radiation, and there forms a bundle less than a centimètre thick, which lies at the level of the second temporal gyrus and second temporal sulcus. A lesion of the parietal or angular lobes induces hemianopsia, only if this bundle be pressed upon directly or indirectly, a lesion of the dorsal part of the optic radiation does not produce a defect in the field of vision. One of Dr. Anderson's, and another of Dr. Bruns' cases, show in what manner the fibres lie in the bundle forming the visual path. The fibres for the dorsal retinal quadrant lie dorsally, and one of my own cases shows that the fibres for the ventral retinal quadrant lies ventrally. It is probable that the macular fibres have a more median situation than the peripheral.

We now enter upon the consideration of the occipital lobe. A number of observations have been made in literature upon occipital lesions. Most of them were accompanied with hemianopsia, but only a few of them are of use in the accurate study of the localisation of the centre of vision. In 20 cases the lesion was diffuse in the medulla of the occipital lobe. None of them localise the visual centre to a circumscribed portion of the lobe, because the visual path was destroyed by the lesion, but many of them go to prove that it does not extend further than the occipital lobe. All cortical or sub-cortical circumscribed lesions, on the contrary, are of great importance in the localisation of the visual centre. The first question that arises is, Does the centre of vision lie on the lateral, mesial, or ventral surface? Eight cases with cortical lesions on both the lateral and mesial aspects of the cortex do not solve this question, but the fact that in 11 cases of lesions situated in the lateral centre, there existed no defect, or only a transitory one, in the field of vision, proves that we need not seek the visual centre in that situation, more especially as, in some cases, the lesion was bilateral. On chart H in my book, I have collected a number of cases without hemianopsia, but with lesions on the lateral or ventral surface of the parietal or occipital lobes, and a look at it ought to convince us that we need not expect to find the visual centre in either of these regions. Just as convincing are the cases with lesions exclusively on the mesial surface (chart G), of which literature mentions at least 25 examples. A methodical analysis of them leads to the following results :—Firstly, a lesion on the mesial surface causes hemianopsia only if the cortex of the calcarine fissure, of the fibres derived from it, are affected. Secondly, a lesion limited to the calcarine cortex can induce a complete hemianopsia, and of this the most instructive case is, I believe, one now published in my book, which includes all guarantees for an exact conclusion. It was stationary, uncomplicated, and the clinical examination, as well as the *post-mortem*, was accurate. The lesion was limited to the cortex in the depth of the calcarine fissure, and the hemianopsia was complete and absolute. Besides, many other negative cases confirm this result, and, in particular, one of my own, with a bilateral destruction of the margo falcata, without defect in the field of vision, proves that the visual centre is not to be looked for there, as Nothnagel and others have affirmed. As to the still more accurate limitation of the visual centre in the cortex of the calcarine fissure, the cases, on the whole, are not sufficiently definite to decide, but there are not at present any absolute reasons for extending it further than the lips of the fissure. In some cases a complete hemianopsia resulted without a lesion in the frontal part of this cortex; in others without any affection of the most posterior part; but in all cases the middle part of the fissure was implicated.

I cannot find in literature any one well-described cases which stands in opposition to the opinion here expressed; but there are many appearing to lead to other conclusions, but which cannot stand a rigorous criticism. Monakow has quite recently arrived at a contrary result. I will not in his absence criticise his theory on a variable localisation of the macula latea, but will only point out my own individual stand-point. I cannot find the clinical cases on which Monakow bases his opinion. He suggests that many spots in the parieto-occipital cortex can, under different circumstances, represent the field of vision. The way in which the fibres of the visual path, pass through the occipital lobe to reach the calcarine cortex, is not clear, but they probably lie in both the dorsal and ventral aspects of the optic radiation. Several facts go to show that, at least, the cortex for the macular field in the calcarine fissure is innervated in a double way.

Organization of the Centre of Vision.—The next question is: How is the centre of vision organised? At first there exists a projection in the calcarine fissure. I have mentioned above that my researches have led me to the conclusion that the fibres of the dorsal retinal quadrant lie dorsally both in the frontal and occipital visual path. The interesting case of Hun's, proves that there is the same arrangement in the calcarine cortex. Thus the upper lip represents the upper retinal quadrant, and other cases strengthen this opinion.

The question now arises: Where does the macular field lie in the calcarine cortex? In deciding this, there are no positive facts, but many cases indicate that the macular field may be more anterior, and the peripheral in the horizontal meridian, more posterior. Some time ago, I observed a case in which the whole of the right occipital lobe was destroyed, and the tip of the left damaged, but still the patient could read up to her death. This case is against the localisation of the macular field being at the tip of the occipital lobe.

Perhaps it is known that Wilbrand, by using purely clinico-ophthalmological facts has come to the conclusion that the macula lutea is innervated by both hemispheres in a variable manner. The clinical analysis of the cases seems to confirm this opinion, and such an arrangement is very favourable to those unfortunate enough to be afflicted with hemianopsia, for they will still preserve the faculty of sight at the point of fixation. As above stated there seems to be another guarantee for the preservation of the macula lutea, and that is, double innervation, both from the dorsal and ventral portions of the optic radiation.

The third important question concerning the cortical retina, as I should like to call the cortex of the calcarine fissure, is whether both eyes or only one are represented in the lips of the fissure, and the crossed as well as the uncrossed fibres. This question is answered by the above case, where a lesion limited to the calcarine fissure had induced a complete hemianopsia of both sides, and others of my cases tend to confirm such a conviction, although it is in opposition to Munk's celebrated experiments.

Of as great interest is the question as to whether the same elements represent homologous points of the retina, or not, and how they are situated in relation to each other. I have myself observed some important facts bearing upon this question, and Wilbrand has elaborated a theory on the subject, supported by some clinical facts. After destruction of both eyes in a patient suffering from leprosy, there ensued a complete atrophy of the calcarine cortex, but in a case of destruction of one eye only, I found in that situation, a number of pigment-charged cells alongside of perfectly normal ones. These facts seem to prove that the elements of both retinal halves are represented in the calcarine cortex by different cells, which lie beside each other. As to the perception of colours, the commonly received opinion that it is situated on the ventral surface will not admit of criticism. Some of my own cases prove positively that colour perception is also situated in the calcarine cortex.

It is well known that this cortex is characterised by a special anatomical construction, and particularly by solitary cells and a layer of small cells, corresponding with the streak of Vicq d'Azyr.[1]

The above expressed opinion in regard to the organisation and limitation of the visual centre to the calcarine fissure does not stand in opposition to the results obtained by the investigation of centripetal and centrifugal degenerations. Monakow also has found by this method that the calcarine fissure stands in connection with certain groups of cells in the external geniculate body. I will not discuss here the complicated problem as to which cells receive the impression of light. It might be left an open question, as well as that of the functions of the different layers in the cortex. After the destruction of the bulbs, or of the external geniculate body, the large as well as the small pyramidal cells undergo essential changes. If the above theory resists criticism, then many of the main problems of the visual path are solved. But we must further exert ourselves to seek new observations, still more accurate and detailed than those hitherto discovered, before the above theory can be considered as finally settled.

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[1] See my work, p. 378.

The next fundamental problem will be to discover the functions of the remainder of the occipital cortex and angular gyrus. But there is no doubt that this cortex has a certain connection with the function of the eyes. Word-blindness, and some other facts in human pathology corroborate such an opinion. I will not comment upon this very complicated problem, which is not yet, in my opinion, ripe for solution.

All these problems are of the greatest importance to psychology, but they cannot be decided without having obtained a solution of the visual centre. Therefore, it seems to me that this study is the most serviceable to us in indicating the right way leading from physiology to psychology.

ON THE PSYCHICAL NATURE OF HYSTERICAL UNILATERAL AMBLYOPIA AND SENSITIVO-SENSORIAL HEMIANÆSTHESIA.

BY PROF. BERNHEIM (NANCY).

At the Congress for the Advancement of Science held at Nancy in 1886, I first demonstrated that hysterical unilateral amblyopia behaves exactly like suggested amblyopia; it is purely psychical. The subject sees with his retina, sees with his brain: the first receives the impression, the second perceives it by its visual cortical centre. But this perceived visual image is unconsciously neutralised by the mind of the subjects of hysterical or suggested amblyopia. They see with the bodily, not with the mental, eye; their amaurosis is but a negative illusion.

I demonstrated this fact by means of a prism and of Snellen's apparatus. We know that a prism placed in front of an eye displaces the corresponding image, and thus produces diplopia. The other eye being closed, there is no diplopia, and the object seen through the prism appears single.

In unilateral amblyopia, hysterical or suggested, a prism placed before the healthy eye should give but a single image, the other eye being supposed to be blind. Now, the patients (who are unacquainted with the theory of the prism) do not hesitate to say that they see two equally definite images, corresponding to the disposition of the prism; hence the amaurotic eye does see.

Stoeben's modification of Snellen's apparatus consists of a pair of spectacles, with a green and a blue glass, and of a black frame, on which there are six letters alternately covered with red and green glass. When one looks to this frame through the spectacles with both eyes open, the six letters are visible; when with one eye only, only three letters are seen, those of the same colour as that of the corresponding glass of the spectacles; the green letters with the green glass, the red with the red. This results from the fact that the mixture of red and green appears black.

Now, our patients, amblyopic through hysteria or suggestion, read all the six letters without hesitation; hence they see with the affected eye; when they close it they see three letters only.

I have shown that hysterical or suggested dyschromatopsia is also purely psychical. Here is a patient with dyschromatopsia of the left eye; a square of green paper are seen grey with it. Now the same square, when looked at with a prism before the right eye, appears double, hence the image formed by the affected eye is green.

Dr. Parinaud, who had first made this experiment, explained it by admitting that achromatopsia existed only in *monocular* vision. He supposes that, in looking with each eye separately, we connect the retina with the opposite hemisphere, but that in binocular vision each eye is connected with both cerebral valves. He has overlooked the fact that the phenomenon is purely psychical. Hysterical achromatopsia is merely a mental illusion concerning the colour of a seen object. The sensorial centre sees the true colour; the mind of the patient does not perceive it.

My explanation has been misunderstood, and it is why I return to the subject. Prof. Pitres, of Bordeaux, after repeating my experiment, says:—"I do not understand how a hysteric can unconsciously mentally neutralise monocular perceptions, without also neutralising binocular perceptions, or, at least, that portion of them supplied by the affected eye. The hypothesis of multiple sensory centres is more satisfactory to my mind, and until further reasons are given, I give it the preference."

My explanation will be clearer if I say that the patient's imagination falsifies or neutralises involuntarily and in good faith the perceptions of one eye, and the following case, in confirmation of this explanation, will disprove the opposed hypothesis.

A youth of 19 fell ill on the 6th of January with gastro-intestinal influenza; diarrhœa and vomiting lasted two days. At the same time there were fits of coughing, ending in attacks of stabbing pains on the left side of the face and body. He also had pain from the xiphoid cartilage to the throat. On admittance into the hospital, the patient, delicate, nervous, indifferently intelligent, was unaware that he was nearly completely hemianæsthetic on the left upper half of the body. The anterior aspect of the thigh alone was anæsthetic and analgesic in the lower limb.

With reference to this hemianæsthesia, let us first examine the amblyopia. The patient cannot read even large print with the left eye, and but vaguely recognises objects. A watch or a hand placed in front of him appear as dark surfaces. He does not see a finger before his eye; when there is nothing, he thinks he can see two fingers. There is also dyschromatopsia. On the 9th of January, white and blue are called red; yellow, blue; and red, grey. On the 12th white is black, and black white; blue is yellow, yellow blue, red grey. These discrepancies in colour vision lead one to assume that the imagination plays a part in the results obtained. The experiment of the prism placed before the right eye shows that in this case also the amblyopic eye sees, for two images of equal clearness are perceived in the respective situations following the disposition of the prism. When the left eye is closed, a single image is seen through the prism by the right eye.

We might suppose with Dr. Parinaud that left amblyopia exists only in monocular vision, but the following experiments disprove this hypothesis. With Snellen's apparatus, the two eyes are armed with red and green glass; the patient sees the six letters and recognises their colours. On closing the right eye he sees everything black. On closing the left eye (red glass), he sees only the green letters, corresponding to the vision of the right eye. This result shows that the left eye sees colours and objects, since he can read the letters without binocular vision, since the right eye cannot see the letters perceived by the left eye only.

One might suppose, it is true, that opening the two eyes is enough to allow each eye to enter into communication with both hemispheres, and that thus the left eye could see with the left hemisphere; but the following experiments show this supposition to be erroneous. After ascertaining that with both eyes open he sees all the letters and their respective colours, that with the right eye only he still sees them all, but perceives nothing with the left eye, he puts on the spectacles. With the right eye (red glass) alone, he reads the red letters; with the left (green glass), he reads the green ones, but does not recognise the colour. Hence the left eye sees objects in thonocular vision, through the right hemisphere, without assistance from the other eye. It sees when the patient's imagination is led astray, and cannot intervene to neutralise or falsify perception.

The following experiment is still more conclusive:—On the 18th of January I send the patient for examination to the eye department. Dr. Koehler, after ascertaining that the left eye cannot read nor recognise objects, says that he is going to examine for selecting glasses, and, with this view, tests the visual acuity by means of Monoyer's scale. He finds that for both eyes it is equal to 0.6; for the right eye, to 0.67; for the left, to 0.5. The left eye alone easily reads the last five lines of the scale.

I repeated the experiment on the 19th, as I could not understand how the patient who was unable to read the largest print on a book or a newspaper had been able to make out much smaller type on the scale. To my great surprise, at a distance of even five metres, he could easily read the five last lines, and clearly distinguish the black letters on a white ground, but at a less distance than five paces he ceases to distinguish, and sees only white strokes on a black ground.

I then try him with colours. Black is seen white (with the left eye) as far as five paces; beyond it appears as black. Like white, blue, and red are seen grey within the limit, but are distinguished beyond. This true vision of objects and colours does not develop gradually, but becomes abruptly manifest at the distance of five paces. The colours and letters of Snellen's apparatus are seen with the naked left eye at five paces and beyond. Within this distance all is black and undefined. With a prism, the left eye alone sees colours at three paces already; the patient, led astray by the prism, has badly calculated the distance; a blue book then is seen blue, but grey at a shorter distance.

These contradictory results can be explained only by the effects of imagination. The mind of the subject, disturbed by the experimental methods; in presence of Monoyer's scale, omits to inhibit the perceived images and colours; and thus the auto-suggestion arose, to see at the distance of five paces. Once surprised into seeing correctly, the eye has kept up the tradition, as it were, and persisted in seeing—but only at the same distance.

All this certainly looks very like a gross imposition. Is it simulation? Yes, if you like; but it is unwitting, compulsory simulation, like that of an hypnotized person.

Both visual fields[1] of the patient are contracted, much more so on the left side. This is the rule in hysteria. The tracing cannot be simulated in a subject like ours, who is quite ignorant of medical matters. He has never been in a hospital, and ignores that hysteria produces such unequal contracted fields of vision. Moreover all hysterical and suggested amblyopias, when examined with prism and Snellen's apparatus, behave like that of our subject. Others, it is true, are more logical and intelligent, do not allow themselves to be too easily taken by surprise, and do not betray the auto-suggestive or pseudo-simulating nature of the phenomenon. But in all it can be shown that the amblyopia appears to be simulated. But then, must we admit that all hysterics combine for shamming unilateral amblyopia, contracted fields, and all the other symptoms of hemianæsthesia?

Let us examine this symptom in our lad. It appears to be complete, tactile, analgesic, thermic, muscular. Pricking, pinching, electrising the left side have no effect. And yet the patient has little self-control; the same stimuli applied to the right side call forth lively demonstrations of pain.

Even when taken unawares the unexpected prick of a

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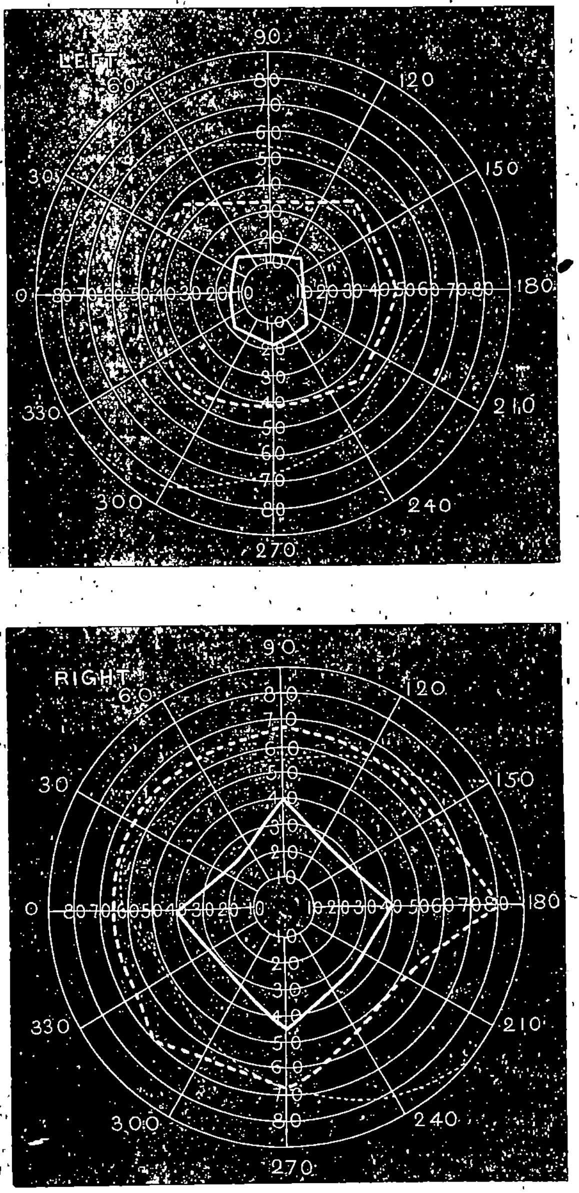
[1] See figs. 1 and 2.



FIGS. 1, 2.

The plain line. Contraction of visual fields on Jan. 10th.
The dotted line. State of visual fields on Jan. 24th, after a suggestion of cure.

pin on the left side leaves him absolutely unmoved. Moreover, the left hand and forearm are bluish and cold, showing the existence of something objective. Some experiments, however, show that there are psychical elements in the mechanism of this anæsthesia. Our patient has thermic anæsthesia, and paræsthesia; a cold object applied to the left hand or forearm appears to be warm, and a warm object cold. On the 15th January I apply his hand to a heated stove; he says it is cold, and leaves his hand in contact, to the extent of burning it, but without manifesting any pain. On opening his eyes and seeing his hand, he says that he feels nothing, neither heat or cold.

This perversion of sensation is possibly but a psychical phenomenon; no physiological explanation can be given. Perverted imagination alone can suggest cold for warm—likewise white for black, *et vice versa.*

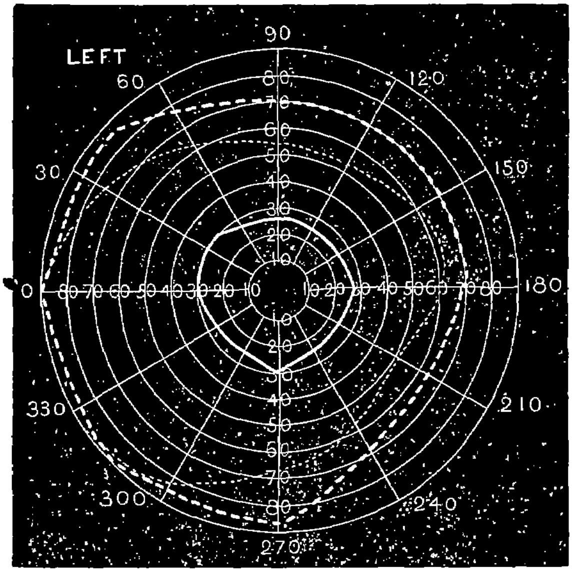
Muscular anæsthesia may also be shown to rest on a mental basis. The patient seems to have lost the notion of the position of his left arm. He cannot pick up a pin with it, nor say whether it is in the air or resting on the bed. With his eyes closed he cannot find his left hand with his right. My own hand placed in it is mistaken for his own right. The patient is hypnotized, and suggestion is made that his right hand is a magnet attracting the left. The suggestion is realised, and this hand goes to be applied to its fellow, showing that the apparent absent notion of its position does exist. After several successful repetitions I explain to the spectators that this experiment shows that the patient feels his hand. Now the experiment fails to succeed. What I have said sets up an auto-suggestion, and the muscular sense is again neutralized. The left hand is still attracted *towards* the right, but ceases to reach it. The patient is roused; he still cannot find his left, and mistakes mine for his. But if I place his own left into his right hand he rejects it as not being his own. He thus was proved to be guilty not of shamming but of auto-suggestion. With reference to auditory anæsthesia, it appears to be complete. He does not hear a watch close to the left ear. One day, however, whilst talking to him, I close his right ear, and continue, quite naturally, to talk to him with a low voice, and from a certain distance. He answers quite well. On being asked how it is that he now hears, he answers that he does not know, that he seems to understand. But from this moment, his attention having been aroused, inhibition sets up, and he ceases to hear the loudest voice. Nay, I then free the right ear and continue to speak in high tones, but into his left ear, as if I was talking for it alone. He does not hear, though the right ear must perceive the sounds. He affirms that he hears nothing. I then talk to his right ear, and he answers. Here again he is surprised, as a victim of auto-suggestion.

The same applies to olfactory anæsthesia. A piece of paper dipped in vinegar and introduced into his left nostril causes neither sensation nor re-action. I push the paper backwards into the pharynx, so that the acetic vapour must affect the right nasal fossa. He continues to feel nothing, though the functions of the latter be otherwise intact.

These experiments prove that in our subject, amblyopia, deafness, anosmia, akinæsthesis, are all purely of psychical origin. Hence, a psychical treatment against the tenacious auto-suggestivity of the patient, gradually restored, in three weeks, all the functions. I allowed one symptom to persist, viz.: the analgesia of the nape of the neck, which still subsisted three months later.

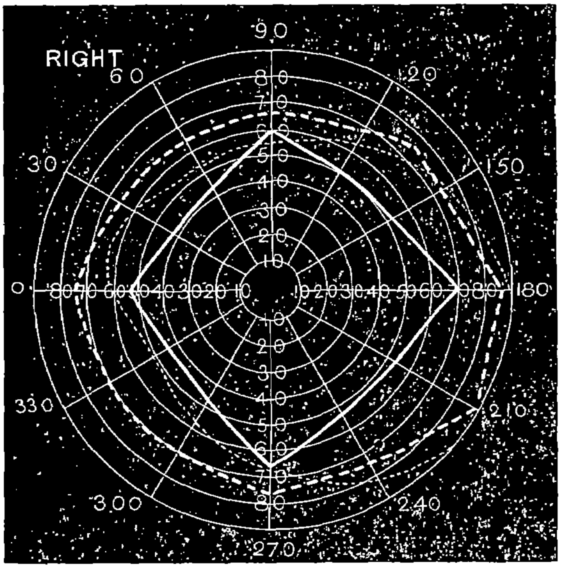
All these anæsthesias, let me repeat again, behave exactly like those due to hypnotic suggestion. But it may be said that hysterical amblyopia presents a great contraction of the visual field, on the diseased, a lesser one on the sound, side. Now I have found it to be the case in unilateral suggested amblyopia. Suggestion made during hypnotism of want of vision in one eye is followed with considerable contraction of the field on that side, and a lesser contraction of the other field. This is shown by the accompanying tracings (figs. 3 and 4).

I do not wish to prove thereby that no organic substratum underlies hysterical anæsthesia. How could a hysteric create by his imagination a left-sided hemianæsthesia? No subject can suggest to himself phenomena of which he has



The plain line. Left visual field after suggestion of amblyopia in this eye

The dotted line. Left visual field after suggestion of cure.



FIGS. 3, 4.

The plain line. Right visual field after suggestion of amblyopia in left eye.

The dotted line. Right visual field after suggestion of cure.

no idea. There must be in his right hemisphere something to set up the symptom. But this something is not in the cortical memory centres. We have seen that they perceive. It must be in the psychical centres, in the cells which are the seat of reaction in consciousness. These cells being modified, it is consciousness of impressions received, which is modified or neutralized. Some disturbance prevents their accepting, or makes them distort, the sensorial impressions from the opposite side of the body. It is a negative illusion, a mental alienation limited to sensations. It is a disease of the conscious æsthesodic cells of the cerebral hemisphere.

ON THE PRODUCTION OF SHADOW AND PERSPECTIVE EFFECTS BY DIFFERENCE OF COLOUR.

BY PROFESSOR EINTHOVEN (OF LEYDEN).

DIFFERENCE of colour may, under certain circumstances, be the cause of an apparent difference in distance.[1] To observe the phenomenon, it is only necessary to glue different coloured figures, such as letters of blue and of red paper, to a screen of black velvet, and to look at them from a suitable distance. In the experiment about to be described, Roman capital letters of about eight by four centimeters were used, the screen being placed at about three metres distance from the observer.[2]

Under these conditions it appeared, both to the writer and to others who he interrogated, that the red letters were nearer than the blue. Obviously, the phenomenon might be explained by difference in accommodation. In order to see the red letters distinctly, a greater amount of accommodation is necessary than in focussing the blue ones, and the greater sense of effort might account for the notion of the red letters being nearer. This accommodation hypothesis, plausible as it seems, cannot however be accepted as a satisfactory explanation of the phenomenon. Several observations tell against it, notably this: that there are about as many persons who see the blue letters before the red, as there are those who see red before blue. In the second place, the apparent difference in distance—so distinct to binocular vision—disappears almost wholly with the closure of one eye. Looked at with one eye only, and for some length of time, the letters appeared to be lying in the same plane, but each time that the other eye was opened, the difference in distance obtruded itself irresistibly.

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[1] Donders. *Wetensch. bijbladen. Med. Gdsth. v. Ooglijders*, 1868.

[2] W. Einthoven. "Stéréoscopie dépendant d'une différence de couleur." *Archives Neérlandaises*, *T.* 20.

The amount of difference remains constant, and can be estimated with considerable accuracy, in the same way as in making a stereoscopical observation. The question therefore suggested itself, whether we had not here to deal with real stereoscopy? The answer to this question is an affirmative. Brücke[1] has shown by means of a simple experiment that the retinal images of differently coloured points are shifted with respect to one another. Looking with one eye at a narrow vertical strip on a black background, the upper and lower thirds of the strip being red and the middle third blue; Brücke observed that the blue part deviated to one side, the two red parts to the other side. By covering either eye alternately, a deviation of the blue and red parts in opposite directions will be observed; and, on both eyes being used, the notion of a difference in distance is proved by the combination of the two images in such a way that the parts that deviate to the nasal side constitute the nearer image; the parts that deviate to the temporal side, the further image. The stereoscopical effect is, however, more distinct and convincing with the coloured letters than with the strip used by Brücke.

The cause of the relative removal of the differently-coloured images lies in the excentricity of the pupil, as may be demonstrated experimentally. The pupils may be made highly excentric by covering them partially. Partial covering on the nasal side is equivalent to a removal of the pupil to the temporal side, and conversely, covering the temporal side is equivalent to removal to the nasal side. With a nasal excentric pupil a shifting of the differently-coloured images in one direction will be observed; with a temporal excentric pupil the shifting will be in the other direction.

The effect of an artificial excentricity of the pupil is surprising when both eyes are used. Anyone who sees the red letters before the blue has only to cover his pupils symmetrically on the temporal side when he will observe that the red letters retreat and soon appear to be behind the blue. On covering his pupils symmetrically on the nasal side, the red letters come forward more and more, and seem at last (experimenting at a distance of four or five meters) to remain several decimeters in front of the blue. A person who sees the blue letters before the red has only to cover his pupils on the nasal side when he will observe that the distances change, the red coming forward and the blue shrinking back.

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[1] Vorlesungen über Physiologie Wien, 1884 3 Aufl. B. 2. S. 95.

Lately, however, Dr. A. D. Waller has written to us that on repeating the experiment with a seemingly slight modification, he obtained the same effects with one eye alone. He used as a test object rings of blue paper on a red ground, or of red paper on a blue ground, and found that the nasal pupil of the left eye gives the same appearance of circular trenches or hillocks as does the temporal pupil of the right eye.

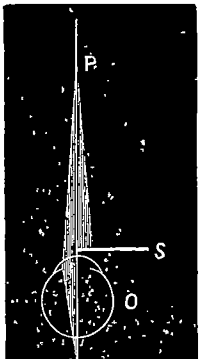
This observation has been the motive to a more thorough study of the phenomenon.

We shall in the first place try to describe the phenomenon exactly, mentioning all such conditions as tend to give it distinctness.

On looking with the right eye and a temporal pupil at red rings on a blue paper, the rings appear as circular hillocks when the paper is held to the left, and also sloping in that direction. One seems to be looking against the dark edges of a thick red ring fixed upon the blue paper. With a nasal pupil the red rings appear as circular trenches.

The phenomenon is the more striking in proportion to the purity of the colours used. The pupil must be made sufficiently excentric and in a suitable direction by means of a black screen that covers it from one side, or better still, by means of a stenopæic apparatus. The pupil must not be too narrow, and the whole eye should be wide open and well directed, so as to avoid any partial covering by the nose, eye-lid or lashes. Lastly, we would point out that it is not desirable to keep the excentricity of the pupil constant for more than a brief period. For if one stares at the rings a long time with unmoved pupils, all appreciation of distance is lost, as in so many cases where only one eye is used, and the rings may even seem to lie in a plane that intersects the plane of the blue paper perpendicularly. If, on the contrary, one shifts the screen or the stenopæic apparatus now and then, the rings appear to rise and sink, and, under the above-mentioned conditions the rising will be with temporal pupil, the sinking with nasal pupil, and in a way almost as striking as if they were seen stereoscopically.

FIG 2.



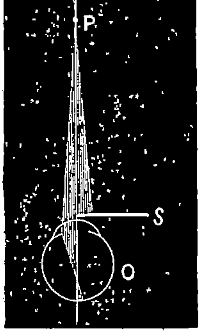


FIG 1.

The explanation of the phenomenon will be found in the appearance of shadows.

If one looks with one eye and excentric pupil at a red and a blue light, one sees the red shifted with respect to the blue, in consequence of the chromatic aberration of the eye. The direction of displacement depends upon the direction of excentricity of the pupil, as is represented by figs. 1 and 2. In fig. 1 S represents a screen which covers the half of the pupil of the eye, and P is a blue light. In fig. 2 S has the same signification, but P is a red light. On the retina red is removed to the opposite, blue to the same side of the screen, so red must be projected outwards to its same side, blue to its opposite side. On looking with one eye at blue and red stripes which are adjacent to one another (see fig. 3) red will be seen removed to *A*, blue to *B*, when the pupil is covered from the side of *A*. Near *m*, the stripes will overlap one another, near *n* and *l* they will be shifted away from one another. A purple border therefore appears near *m*, having an intensity of light equal to the sum of the intensities of a red and a blue stripe, whilst near *n* and *l* dark borders will be seen.

If the screen covers the pupil from the side of *B*, *l* and *n* will be light and *m* dark.

What has been said of the coloured stripes also holds good, of course, of the red rings on a blue ground, or blue rings on a red ground; they show light and dark borders in the same way when looked at with one eye and an excentric pupil.

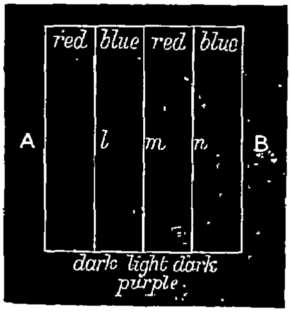


FIG 3.

Leaving for a moment the colours, we must now treat of the shadows that are seen when there is a small but steep depression or elevation on an equally lighted (uncoloured) and flat ground.

Our first enquiry is as to the apparent illumination of the flat ground and of the edges.

If the light falls in parallel rays and perpendicularly to the ground, the steep edges *a* and *b* (see figs. 4 and 5) will remain dark. If the light falls obliquely, they may receive either more or less illumination than the ground. If the light falls slopingly from the side of *A* (see fig. 4) or from the sides before or behind the plane of drawing, *a* will remain dark with all degrees of inclination of the rays. And even when the rays proceed from the fourth side *B*, *a* will receive less illumination than the ground, if the inclination of the rays is more than 45°. Only in the case of the rays proceeding from *B*, and at a slope of 45°, or less, will *a* be as much or more highly lighted than the flat ground.

In the same way an edge, *a* or *b*, in fig. 5 will, in by far the greater number of cases, be darker than the ground.

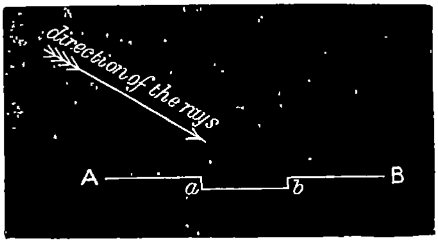


Fig 4.

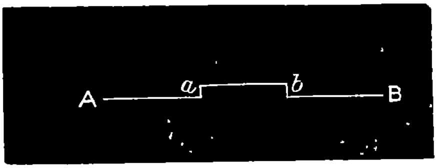


Fig 5.

We now proceed to give a mathematical treatment of the subject. In the midst *M* of a sphere (see fig. 6) imagine a flat ground with a small perpendicular edge, just as in fig. 7, and imagine the surface of the sphere infinitely large with respect to the surface of the ground. The latter lies in the horizontal large circle *ACBD*, the edge $v\ v_1$ (fig. 7) lies in the vertical large circle *CFDE*.

Let us suppose that the rays which fall from a single direction are reflected diffusely, so that the intensity of the reflected light, or the apparent illumination, depends only on the strength of the incident light and the angle of incidence.

Let us ask, in the first place, what chance there is of the flat ground being more highly lighted than the rising edge when the rays come from a single direction and all directions are equally probable?

Construct the large circle *CHDG*, which intersects the rectangular biangle *ECDB* in the middle, and from the point *P*, arbitrarily taken *below* that circle on the surface of the sphere, draw a ray *PM*, and then draw PP_1, a perpendicular on the plane *ACBD* and PP_2, a perpendicular on the plane *CFDE*. If *PM* represent the intensity of the incident light, PP_1 and PP_2 will represent the respective intensities with which the flat ground and the edge are lighted.

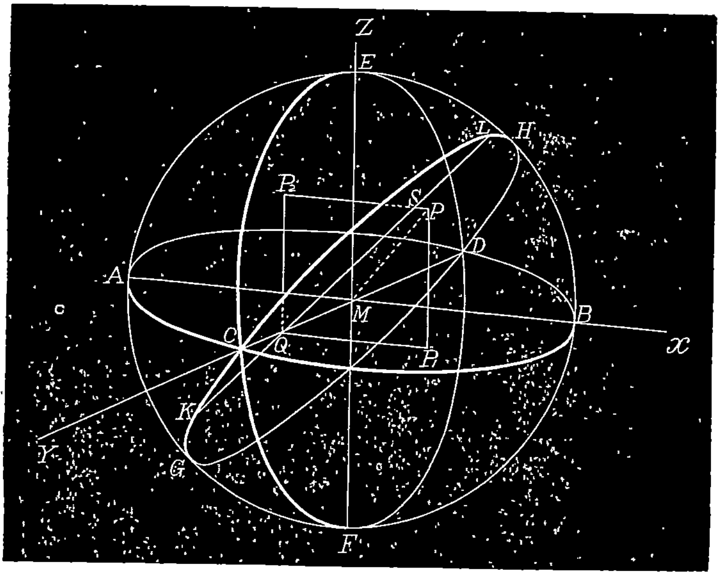


FIG 6.

Lay a plane through PP_1 and PP_2, this intersects the horizontal large circle *ACBD*, according to the line P_1Q, and the perpendicular large circle *CFDE*, according to the line P_2Q. PP_1QP_2 is a rectangle, which is intersected by plane *CGDH*, according to the line *QS*. The line *QS* cuts the angle P_2QP_1 in the middle. As the point *P* is taken *below* the plane *CGDH* on the surface of the sphere, the point of intersection *S* must lie between *P* and P_2, therefore PP_2 is longer than PP_1, *i.e.*, the edge receives more light than the ground. If *P* were lying *in* the circle *CGDH* on the surface of the sphere, the apparent illumination of both planes would be equal; if it were lying *above* that circle, the ground would receive more light than the edge. The chance of the latter being the case is expressed by the proportion of the curved surfaces of the spherical biangles *ACDH* and *BCDH i.e.*, = 3 : 1.

When we take into consideration that, in order to still be able to see the ground, the quantity of light which is reflected by it, cannot be diminished below a certain value, it is obvious, that *P* is to be taken at a certain distance from the horizontal circle *ACBD*, and this increases the chance of the ground being more highly lighted than the edge.

Let us ask in the second place as to the illumination of the horizontal and of the perpendicular plane, when the rays fall from all directions at the same time and with equal intensity. It is easily understood that in this case the horizontal ground will be lighted by the surface of half a sphere, whilst the perpendicular edge receives rays from only a quarter of the sphere-surface; the proportion of the apparent illumination will therefore be =2 : 1. In reality the illumination of a ground will in most cases be a combination of stronger light from one side with weaker light from all other sides; therefore, the chance of the ground being more highly lighted than the edge—already greater than 3 : 1., with illumination from a single side—will under ordinary circumstances be considerably increased. Generally, therefore, an observer looking at the steep edge of a depression or elevation on a flat

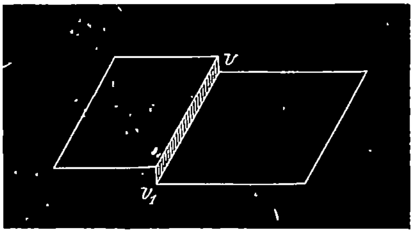


Fig 7.

ground, will see this as a dark shadow. On the other hand, under certain circumstances, he will involuntarily conclude merely from the appearance of a dark border, that he is looking at a steep edge.

That this is the case, when under the above conditions, one looks at red rings on a blue ground or blue rings on a red ground, and sees them as hillocks or trenches, can easily be proved by looking at the rings, first from one side, and then from the other, keeping the pupil excentric in the same direction. Experimenting in the way described on p. 193, and looking at red rings on a sheet of blue paper with the right eye and temporal pupil, the red is apparently removed

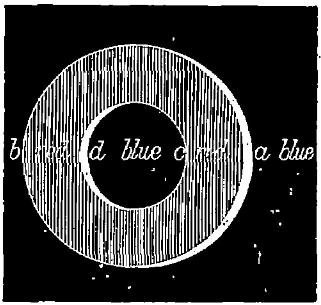


FIG 8.

to the left, the blue to the right, the ring showing a shadow on its outside at the right (fig. 8 *a*) and on its inside at the left border (*d*).

The sheet of paper was held to the left side, sloping in the same direction, and the red appeared to lie upon the blue. *On removing the sheet of paper to the right, and making it slope in that direction, the shadows will be formed with the same temporal pupil on the same places, but they are now looked at from another direction. Therefore, instead of rising above the blue, the red will appear to sink* (*figs.* 9 *and* 10).

If the sheet of paper is held on the same side sloping in the same direction, the left eye will see with nasal pupil what is seen by the right eye with temporal pupil; for the pupil is not to be removed to the symmetrical, but to the same side, in order to shift the colours on the retina in the same direction

With pupil excentric in an upward or downward direction you have only to hold the coloured paper straight before you, and sloping forwards or backwards in order to cause the same illusion; and the effect may be obtained equally well with the pupil excentric in all other directions, provided the sheet of paper be removed in the same direction or the opposite one, and that it turn on an axis which crosses that direction perpendicularly and lies in the plane of the paper.

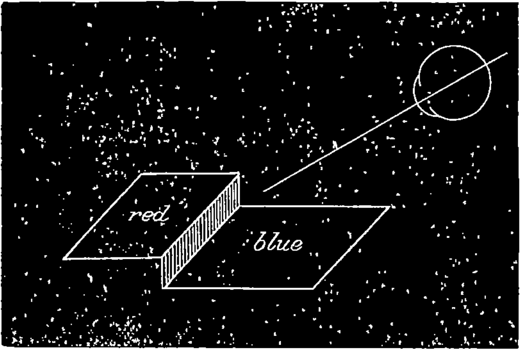


FIG 9.

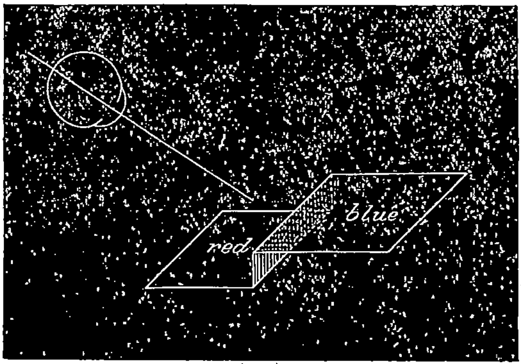


FIG 10

As is mentioned on p. 195, purple-coloured light borders are observed on the rings near the dark borders. When they do not catch the eye too much, they seem to confirm the illusion by antithesis. On experimenting with a half-red, half-blue coloured paper, a single straight line of demarcation was shown, and red appeared before blue or blue before red, under the same conditions as with the coloured rings, when there appeared a dark border; when, however, a light border was seen no striking difference in distance could be noticed.

We have still, in conclusion, to treat of the question in how far the shadow that is thrown on the ground by a rising edge can affect the illusion mentioned. And here it must be taken into consideration that the light falls only in two directions, one opposite to the other, as when experimenting with the coloured rings we only see shadows lying either towards us or in a direction straight away from us.

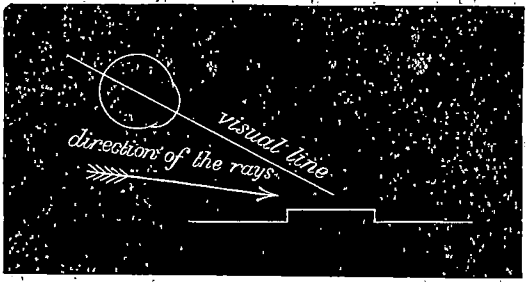


FIG 11.

If the light proceeds from the one side, the shadow that is thrown will enlarge the dark stripe of the rising edge, and thus strengthen its impression. If the light falls from the other side the shadow thrown will ordinarily be invisible to the eye of the observer.

An exception to this occurs with such position of the eye and direction of rays as is represented in fig. 11, where moreover the edge is more strongly lighted than the flat ground. We investigated the manner in which the coloured rings were judged as hillocks or as trenches under these conditions. The experiment can easily be performed. We did it by means of direct sunlight, and found that the trenches, as well as the hillocks, were formed in the same manner as with the usual light.

We thus learn that notwithstanding that we know in a particular case the exact direction of the rays and the spot where, under these given conditions, the shadow *should be* formed, yet a visual impression is judged as if the shadows were caused by rays falling in the directions of most frequent occurrence.

ON ATTACKS OF LETHARGY AND ON HYPNOTIC SUGGESTION.[1]

BY PROFESSOR HITZIG.

ALTHOUGH, on the whole, I am not very fond of casuistics, I beg leave to call attention to a single case of hysteric sleep, which I have successfully treated by the employment of hypnotic suggestion. I do so, not only to submit this method of treatment for criticism, but because the case appeared to me to be not altogether without value on account of its ætiology, the peculiarity of its course, and on account of some observations which were made on the metabolism before, during, and after the sleeping fits.

A labourer, twenty years old, met with an accident in the night of the 11th of July, 1891, working a dredging machine. A bucket became loose and fell on his left arm, and a connecting-rod on the left side of his head. He sustained some considerable wounds of the skin and muscles of the extensor side of the left fore-arm, and some minor cuts on the left side of the head near the middle line. General symptoms were wanting; indeed, he was able to call upon the doctor himself.

He was further treated in a hospital, where the wounds on the head healed in four or five weeks, and those on the arm in about twelve weeks.

About the middle of August, according to the statements of a medical report, at intervals of from two to three weeks: and, according to the statements of the patient, at first every eleventh or twelfth day [subsequently every Tuesday or Wednesday], *states of excitement* began to set in, accompanied by headache and *rise of temperature*, upon which, as soon as the latter had reached 39 C, *a period of sleep* regularly followed. The sleep usually lasted from the evening of one day till noon of the third day, so that the patient slept nearly two days. The increase of temperature, which at the beginning of the attacks was said to reach 39.9,° become later less and less marked, until the temperature during sleep was stated to amount to only 36.3° C.

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[1] Read before the Congress of Experimental Psychology, London, 1892.

During the sleep complete amnesia prevailed. During that time the patient never took any food, but during the attack he repeatedly drank, and discharged urine. The nurse, it was said, pressed, for this purpose upon the scar on the arm, whereupon the patient became awake for a few moments, and asked for a drink. Even this, however, he only learned from the lips of others.

After the attack regularly, but only for a short time, great faintness prevails, and a violent headache; after that the patient begins to feel very well, and to take abundant food.

Present State.

The patient, a middle-sized, strongly built and well-fed man, exhibits on the left parietal bone, about 3 cm. from the middle line, some movable scars, which are not painful when pressed. But another spot, of the size of a finger-joint, is painful when pressed, not so much when tapped; it is situated about the breadth of three fingers to the left of the sagittal suture behind the other scars, just midway between the coronal and lambdal sutures.

On the dorsal aspect of the left forearm there is a large red scar, forming an irregular acute angle; transversely it measures 8 cm., and reaches from the medial edge of the ulna obliquely upward to the middle of the radius, its longitudinal portion, 5.5 cm. long, lying above the radius. Across the middle of the fore-arm, corresponding to the scar and below it, there is a deep cavity caused by muscular defect; it deepens when the muscles are contracted, and at the same time the transverse portion of the scar is displaced with the muscles. On the dynamometer, the patient presses 55 kg. with the right hand, 21 kg. with the left.

When the face is not moving, the left naso-labial furrow is much more marked than the right one. When one-sided movement takes place, this difference is also clearly to be seen, and much more so when bilateral movement reaches its maximum. *Sensibility* is altered only near the scars, so that touches with a brush and pin-pricks are felt more painfully about a finger's breadth above them, and about 7 cm. laterally and below. Pressure on the transverse portion of the scar, and on a spot immediately below the middle of it is very painful, and it produces abnormal sensations in the back of the head. All this region is very sensitive to the touch of the shirt, and there is slight left-sided ovarian tenderness.

The knee-jerk is increased, and also produced by a blow on the tuberositas tibiæ.

Other anomalies are wanting.

When I saw the patient, for the first time, on the 15th of February, 1892, I told the assistant physician, with a suggestive intention: "You will see that the patient instantly falls asleep as soon as I look in his eyes." In fact, after I had fixed his eyes for a few seconds, I had put him into the hypnotic sleep. Afterwards hypnotism, easily produced by any means, was usually effected by the humming of a deep tuning-fork.

In his hypnotic state a complete left-sided hemianæsthesia was suggested to him for clinical purposes. Therefore, when I presented him in the *Clinique* on the 24th of February, he was completely anæsthetic all over the left side of his body to all sorts of stimulation, even the strongest induction currents. He had besides lost the sense of smell in his left nostril, and that of taste in the left side of his tongue. His organ of hearing had not been affected, but I suggested to him in a high degree a concentric restriction of his field of view in his left eye, and in not so high a degree in his right one. I succeeded in doing so, first by shrinking in a high degree both fields of vision, and then by widening the right one, as far as I pleased. So he was, on the whole, a specimen of a hemianæsthetic patient, as it is principally known to us by Charcot's classic descriptions.

After the demonstration, I made him fall asleep, and, by a short verbal suggestion, I made the whole hemianæsthesia disappear again before the clinical students.

The hypnotic state was made use of for some other ends besides. First of all, I thought it useful to render *anæsthetic* once for all, *the painful scars* on the arm of the patient. It is well-known since the publication by Pitres that painful spots on different parts of the body may play the part of so-called "points hypnogènes," which I need not explain here more in detail. Though simple stimulation of the scars did not produce any fit of sleep, still a relation between the scar and the fits is made probable, for the patient, who did not react against other stimulation, could be awakened for a short time by pressing the scar. So, for my part, I am of the opinion that scars are much more commonly causes of hysterical than of epileptic attacks. I, therefore, wished to exclude from the sensorium the painful stimulation, which was produced by irritating the scars. I placed my confidence in the effect of anæsthetizing by suggestion; but isolated anæsthetizing of the scars miscarrying, I was obliged to anæsthetize the whole fore-arm. The effect of such a suggestion done but once, lasted for several months; so far completely, that the patient very soon did not perceive a touch at all, and painful stimulations he only felt as a touch. I do not think it impossible that the diminution and the temporary suppression of the prodromal loss of weight, of which I shall speak further on, is to be explained in the same way.

Hypnotic suggestion was next employed against prodromal headache, partly with complete, partly with, but passing success.

Lastly, I employed it against the fits of sleep. However, before discussing this application of the treatment, I shall describe the course of the fits of sleep themselves.

The patient had finished his last period of sleep out of the clinique, exactly eight days before the setting in of the first fit, which attacked him in the clinique. On the whole, he had seven fits in the clinique; each of them was preceded by a prodromal stage.

Usually, about two days before the setting in of the sleep, the weight of the body began to decrease. At the same time, pain and humming in the head began; the expression of the face, cheerful and open before, became distrustful and irritable; in a similar way, he was mentally changed, and the nearer the fit approached the more his eyes closed, so that one could judge from the patient's eyelids hanging down that a fit was approaching.

The fits of sleep, except the last one, which was stopped, lasted from 34 to 36 hours, once 41 hours. *The intervals* lasted from 7 days and 13 hours to 13 days and 13 hours. *During the fits* the patient usually reclined quietly on his back; sometimes, however, he tossed about. This principally took place after 15 cgrs., churrùs (a very active species of cannabis indica) had been given to him during his fit. The eye-lids did not quiver. Contractions or convulsions, especially of the masseters, were not present. The eyeballs were directed upward, and when the lids were kept open for a time, slowly moved to and fro. His temperature, pulse, and respiration did not show any considerable changes; the last two only when churrus had been given.

The patient could be awakened by any means, sometimes, by pressing the left ovarian region or the scar; then he asked for water, drank and passed urine, whereupon he at once went to sleep again. Once, on the 2nd of March, he awoke spontaneously and behaved in the same way.

During his sleep, the weight of the body, the volume, the specific gravity and the quantity of nitrogen in the urine decreased. The secretion of phosphoric acid was not examined.

After his sleep, the patient had a more or less violent headache; soon, however, he began, with a good appetite, to eat freely; the weight of his body rapidly increased; in the same way, the volume, the specific gravity, and the nitrogen of the urine increased. The patient then felt very well, worked hard indoors and in the garden, and betrayed no signs of a hysterical character till the prodromata of the new fit began to act.

I said above that the patient once had a long interval of 13 days between his fits. It lasted from the 21st of March till the 3rd of April. It is to be noticed that during this time, *i.e.*, on the 25th of March his usual prodromata took place, and on the 27th of March very free bleeding of the nose occurred. Then he felt as if he had gone through his sleep, and, indeed, between the bleeding of the nose and the sleep, there was the same mean interval as usually took place between two sleeping periods. In this case, evidently, the bleeding which interrupted the regular course of the symptoms took place as an equivalent of the fit. Then I tried suggestively to interrupt this course in different ways, supposing that if this end could be really attained, the fits would be effectually stopped. For this purpose, I hypnotically suggested to the patient several times in the interval that the fits would leave between them a pause longer than usual and would not reappear before a certain day appointed by me. Then I suggested to him during the sleeping fit that the fit would last a shorter time than usual, in fact, till an hour appointed by me. These experiments, however, had no result whatever.

Then I hypnotized the patient on the 26th of April, in the afternoon, during the prodromal stage, when he had a violent headache, and when from his half-closed eyes it might be with certainty supposed that his lethargic sleep would occur that night. Then I suggested to him that I had just put him into a magnetic sleep, that this sleep never passes into his morbid sleep, and that though I had not been able to awake him from his morbid sleep, I should awake him from his present sleep. This would take place on the next day. I should three times clap my hands, the third time he would awake, but not before. In this way he would be cured; he would never again suffer a new fit.

I adopted this process from reasoning that, since experience shows that the effect of suggestion usually (as was, in fact, the case of our patient), decreases on account of other sensations intervening, suggestion ought to have an extraordinary effect, if it were made as near as possible to the impending fit, if it were applied during the hypnotic state, and if by inducing the hypnotic state external stimuli were excluded, as much as possible during the interval. As a matter of fact the result agreed with this reasoning.

On the 27th of April, 22 hours after the setting in of the hypnotic state, the patient awoke when I clapped my hand for the third time. Since then, that is, during more than three months, he has never had a fit. Nevertheless, the patient is by no means completely cured. His prodromal symptoms described above still occur, but they appear, it is true, in irregular and longer intervals, of from a fortnight to three weeks, and his head-ache is not so intense as before.

These symptoms still usually last two days, as before, and they are accompanied with the former irritability and excitability of his state of mind. The patient says that though he remembers all the events of those days, yet he afterwards fancies that he did everything in a dream. Besides this, the scar and its neighbourhood always becomes sensitive, as soon as I remove the anæsthesia of the fore-arm.

In this case we again draw the conclusion that, by suggestion, we can remove some hysterical symptoms, but that by it alone we are not able to remove the hysteria itself. The patient has been submitted to a therapeutic treatment, but it has so far not proved completely successful.

Now, I desire to call attention to some points of very particular interest. First, as regards the character of these pathological states of sleep, I am of opinion that, from the recent investigation by Charcot and his school, above all by Gilles de la Tourette and Cathelineau, by Lowenfeld and others, there can be no doubt but that they belong to hysteria. Therefore, I do not hesitate to include the fits of our patient among hysteria, though, in this case, not only all the symptoms of hystero-epilepsy were wanting, but also all the other symptoms of hysteria, except the ovarian, the susceptibility of being hypnotized and the possibility of suggestion. From this very reason the case seems to me to claim a greater interest, because differing from the majority of the cases hitherto published. It gives a very clear idea of lethargy occurring in a marked degree, and modified only by our various operations upon it. Further, agreeing as I do with the doctrine of Charcot and his followers, I am inclined to consider the sleeping fit as a part of the hystero-epileptic attack, and in our case, therefore, as an abortive fit. Not only the general impression, the prodromata, the fit and the post-hysteric state strikingly remind us of the analogous states of epilepsy and hysteria, but, in our case, there is also a certain, though not complete, confirmation in the very peculiar changes of nutrition with those in the fits of *grande hystérie*, which according to the investigations by Gilles de la Tourette and Cathelineau, are identical in the various other phases of the hystero-epileptic fit and the sleeping fits.

In the second place, the ætiology of the disease is full of interest. Without any doubt we have to do here with a functional neurosis of hysteric character caused by a trauma. It is not in the least my intention to enter into the dispute on traumatic neurosis which of late has been so vigorously conducted. Suffice it to say that I have seen a very considerable number of traumatic diseases of the central nervous system of a functional character, and that they may be divided without any difficulty into hypochondriacal, neurasthenic and hysterical forms. The latter, though not so common as the other forms, are not at all rare, considered by themselves. Therefore, I do not regard with approval the view of certain authors who, because they have not observed such cases, or because they did not recognise them, declare the occurrence of hysteria and hypochondria in persons of low extraction, above all in males, and especially after trauma, to be exceptional, and who assert that other authors, who in this respect command a greater experience, have been deceived by dissemblers. The nutritive processes before, during, and after the sleeping periods, seem to me particularly worthy of the attention of scientific men, because their study may be presumed to afford a nearer insight into the changes of the cerebral mechanism occurring in the fits of *grande hystérie*.

The few facts I can impart in this respect differ in some points from the observations of Gilles de la Tourette and Cathelineau. These authors have stated that the nutritive troubles described by them did not begin before the fits set in, and ceased with them. This did not apply in my case. Before and during the first two fits, the patient lost 4 and 3 klg. respectively. I cannot tell how much of the loss is to be put to the account of the prodromal period, because daily weighing was not then made. During the prodromata of the three following fits, however, he lost 2 kg., 750 g., 2 kg. and 1 kgr. Before the sixth fit no loss of weight was to be detected, and before the seventh fit, which was the last, it amounted only to 500 gr.

You will have noticed that these figures, except the last, show a continual decrease, and I once more insist upon the possibility of the improvement being caused by the diminution of the prodromal headache, perhaps as well by suggestive anæsthetisation of the scar. There is, however, an inverse ratio between the decrease of the weight of the body during the fit and the decrease before the fit; the latter more and more diminishing, the former augmented in the same degree, so that the addition of these two figures always yielded the same weight during the whole of a fit, that is twice 3 kgr. 500 gr. and twice 3 kgr. 250 gr.

Not only the weight of the body, but also the excretion of nitrogen showed considerable changes during the prodromata.

In order to be surer of my ground, I desired Professor von Mering to ascertain the nitrogen, which he did according to Kjeldahl's method. Unfortunately, the results furnished me did not form a complete series, so that I must confine myself to stating them simply. Thus, the secretion of urea increased from 24.6 gr. on the day before the setting in of the prodromata of the fourth fit to 30.21 gr. during the first day of the prodroma, and to 44 gr. during the second. The patient having headache on these days, and therefore certainly not having consumed more food than usual, but having lost considerably in weight, the augmented excretion of urea may with certainty be put to the account of the loss of weight.

Easier to explain is the augmented excretion of nitrogen immediately after the fit, which, in three fits, reached its highest point with 37.95, 33.25, and 30.4 gr. of urea, on the day after the fit, for it has been ascertained that on these days the patient consumed a much larger quantity of food. During his sleep, as has been stated, the secretion of urea diminished, corresponding to the statements of Gilles de la Tourette and Cathelineau. The lowest standard noticed during a complete sleeping day amounted to 5.93 gr.; the patient, at the same time, ate nothing, and sustained a considerable loss of weight. The latter, therefore, may be put (or to a large extent) to the account of the metabolism destroying tissue. The volume of urine generally fluctuated very much, but during the five fits in which it could be measured, it showed, relatively speaking, great regularity. It was diminished, and reduced on the basis of 24 hours, amounted to 1000, 987, 770, 867 and 800 ccm. In the last fit part of the urine was lost.

Of course there was, during the intervals between the fits, an inverse ratio between the specific gravity and the volume of the urine, and it was, therefore, also very fluctuating. But on the complete sleeping days it was very constant, that is, 1017, 1018, 1016 and 1017. On the sleeping day, which had been ushered in by the hypnotic sleep, it amounted to 1020.

I am very far from drawing far-reaching inferences from the few observations which I have given above. I think, however, I am justified in saying that the question of the nutrition during the hysterical sleep is not yet answered by the formula.

"Little metabolism occurs in hysterical persons during the fit": this formula contains only a part of the whole truth. Finally, since many command a much more extensive store of observations on hysterical persons than myself, I beg to call attention to the mode of cure which I applied. I am far from thinking that persons with inveterate hystero-epileptic fits can be cured by anæsthetizing hysterogenic zones, or by stopping their sleeping fits by means of hypnotising and suggesting at the same time. But since every outbreak of inveterate fits is preceded by a period more favourable to our therapeutic operations, the experiment of leading the disease into another channel during this period, in the manner I have described, seems to me worthy of trial.

Month	SLEEP.		INTERVAL.		Loss of weight during sleep.	Loss of weight before sleep.	Increase of weight after sleep.
	Days.	Duration.	Days.	Duration.			
February.	9th ev. to 11th noon.	40 hours.					
February.	19th 2 p.m. to 21st 8 a.m.	42 hours.	11th noon to 19th 2 p.m.	8 days.		4 kgr.	5.5 kgr.
March.	1st 3 p.m. to 3rd 7½ a.m.	41 hours.	21st Feb. 8 a.m. to 1st March 3 p.m.	9 days 7 hours.		3 kgr.	3.5 kgr.
March.	10th ev. to 12th 8 a.m.	34 hours.	3rd 7½ a.m. to 10th ev.	7 days 14 hours.	0.750 kgr.	2.750 kgr.	5.5 kgr.
March.	19th 9 p.m. to 21st 8 a.m.	35 hours.	12th 8 a.m. to 19th 9 p.m.	7 days 13 hours.	1.5 kgr.	2.0 kgr.	6.0 kgr.
April.	3rd 9 p.m. to 5th 8 a.m.	35 hours.	21st 8 a.m. to 3rd 9 p.m.	13 days 13 hours.	2.250 kgr.	1.0 kgr.	2.5 kgr.
April.	13th 9 p.m. to 15th 9 a.m.	36 hours.	5th 8 a.m. to 13th 9 p.m.	8 days 13 hours.	3.250 kgr.		3.0 kgr.
April.	26th noon.		15th 9 a.m. to 26th.	11 days.	2.0 kgr.	0.5 kgr.	3.750 kgr.

ON DIFFUSE ENCEPHALITIS: WITH AN ACCOUNT OF A CASE [1] IN WHICH THE PATIENT SURVIVED.

BY R. LAWFORD KNAGGS, M.C.

AND

R. CONYNGHAM BROWN, M.B.

JAMES C——, age 10, was admitted into the Huddersfield Infirmary, on October 3, 1891, under the care of Mr. Samuel Knaggs, for spastic paralysis of the left arm and leg and suffering from fits.

He was the son of healthy parents, and one of a healthy family.

He had perfect health till five years before, when he fell from a wall seven or eight feet high. He was taken indoors, and, though apparently none the worse for the fall (?) it was thought advisable to put him to bed. The following morning he could not be roused, and a medical man was sent for, who "said he was suffering from compression." The greater part of that day he lay unconscious, and then gradually recovered; and in a few days was going about as usual. A month later, without any apparent cause, he fell down in a fit; he was unconscious, and "twitched his left arm and leg." After an interval of two months he had a similar fit, and, for a period of two years, the fits recurred every other month. Between them the boy appeared all right, but for slight loss of power on his left side, which was noticed towards the close of that time.

Nearly three years before admission he had a second fall; and though it was only from a height of two or three feet he was taken up insensible. For two weeks he lay unconscious and perfectly quiet, the only change being slowly increasing emaciation; and then, though gradually regaining strength, he was unable to rise from his bed for eight months.

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[1] The writers are indebted to the kindness of Mr. Samuel Knaggs for permission to publish this case, and to Dr. F. L. Mackenzie for some valuable notes about the early history of the illness.

Dr. F. L. Mackenzie, who was called in to see the patient somewhere about this time, has kindly supplied us with the following notes of his condition. On January 9, 1889, he saw the patient for the first time, and "found him in an extremely weak and emaciated condition. He lay on his right side, his eyes half open, and without any other characteristic facial condition. He was coiled up, his head being flexed upon his chest, and both thighs acutely flexed on the abdomen. When shouted at, or shaken somewhat roughly, he responded with a howl and again became quiescent. There was no evidence of hemiplegia. The emaciation was so extreme that unilateral wasting could hardly have been detected. His pupils were equal, and only slightly responded to light. The conjunctival reflex was very imperfect, and so was the knee tendon reflex." "Urine and fæces were passed in bed. Respiration was regular, and not indicative of cerebral disease. P. 120, small. Temperature 97.8." "In the course of a week the patient began to move in bed, and take more nourishment."[1] By the middle of April, his general condition was greatly improved, and he was moderately intelligent, but "as movement of the limbs returned, evidence of hemiplegia showed itself—flattening of the left lip, slight contracture of the left hand, and flexion of the left leg" (which last was corrected by extension). The condition of spastic paralysis, which the arm presented on admission, gradually developed, the flexion being first noted at the wrist, and then at the elbow.

During the eight months the patient was in bed, he had no fits, but shortly after getting up he had one, in which he is stated to have squinted and worked the left side. These fits continued with increasing frequency till his admission. Dr. Mackenzie was again consulted on September 7, 1889, and describes "attacks of sudden jerking, especially of the left leg"—"a sudden clonic spasm sometimes causing the patient to fall." "There was no loss of consciousness, no aura and no drowsiness after the attack." At first, these attacks occurred two or three times a week, but they practically disappeared under treatment by the end of November. In March, 1891, Dr. Mackenzie saw the boy again for "similar attacks affecting the upper extremity more markedly than formerly." They occurred now as often as two or three times a day, and treatment had no effect upon them.

With the increase in number of the fits, the loss of power on the left side became more marked, and for 18 months previous to admission his parents stated that he had become peculiar in manner and difficult to control.

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[1] Mercury was given during this period, and its value seems to have been very marked.

On admission (October 3, 1891) the patient was a powerfully-made well-nourished, healthy-looking boy. His bright eyes gave him an intelligent expression, but his slowness in answering questions, deliberate enunciation, and eccentric behaviour evidenced an impaired intellect.

His gait was peculiar and jerky, owing to the spastic condition of the left leg, which was advanced without bending the knee. The jerkiness was communicated to the left arm.

There was slight left facial paresis and a spastic paralytic condition of the left arm and left leg.

The left arm was always held close into the side. The hand was bent at right angles to the fore-arm, and the phalangeal joints were flexed. The fore-arm was pronated and bent almost to a right angle with the arm.

The hand was smaller than the other, and the limb slightly thinner than the right.

The deltoid was particularly flabby. The arm could be abducted nearly to the horizontal by rotation of the scapula, but the deltoid was inactive. With effort he could partially extend the elbow, but, once extended, it was flexed without difficulty. There was hardly any power of movement in the wrist and finger-joints. There was considerable rigidity to passive movement; which was most marked in extension.

The left leg was extended. It was of equal length, but slightly thinner than the right, and the power of voluntary movement was only slightly inferior. The greatest difficulty was shown in extending the foot.

Rigidity to passive movement was present in flexion and extension, and especially in abduction.

On the left side there was slight ankle clonus and an exaggerated knee jerk. Sensation was normal on both sides. The fundus in both eyes was healthy, and no evidence of an old papillitis could be detected.

The heart, lungs, and abdomen were normal, and the urine had a sp. gr. 1020, and contained no albumen.

The boy was kept under observation for a fortnight, during which time he had at least three or four fits every day. They were of an epileptiform character, and showed a distinct tendency to become more frequent.

He had a stiff feeling in his left arm when they were coming on. During the fit he became unconscious and fell. The mouth was drawn to the left side, and a stiffening of the left arm and leg was succeeded by clonic spasms; these, though most marked on the left side, were never entirely limited to it. A fit lasted about two minutes, and on recovering he became pale and faint for a few seconds and then said he felt well again.

The convulsive movements did not begin in any particular muscle or group of muscles. At other times the left arm would be involuntarily jerked forward once. This was of frequent occurrence and was unaccompanied by any other symptom. The temperature was normal, but when taken in the left axilla it usually registered nearly a degree lower (subnormal) than when taken in the right.

The history, and the unilateral and progressive character of the paralysis and of the convulsions pointed to some lesion of a traumatic character implicating the R. motor area, and after consultation it was decided to explore over that part of the brain.

Operation. On October 16 (the boy's head having been previously shaved and rendered aseptic), the situation of the fissure of Rolando was marked out on the right side of the scalp, a large semi-lunar flap reflected down, and two half-inch discs of bone removed above and below a point situated over the arm centre. The dura mater did not pulsate very evidently at this stage, and it did not bulge. A third disc in front of, and intermediate between the other two was removed, the intervening bone marked out by Hey's saw, and clipped away with bone forceps, and in this way a triangular aperture was made whose sides measured from 2 to 2½ inches. The exposed dura concealed the lower ⅔ of the fissure of Rolando, and the centres for the face and arm, and part of the leg centre.

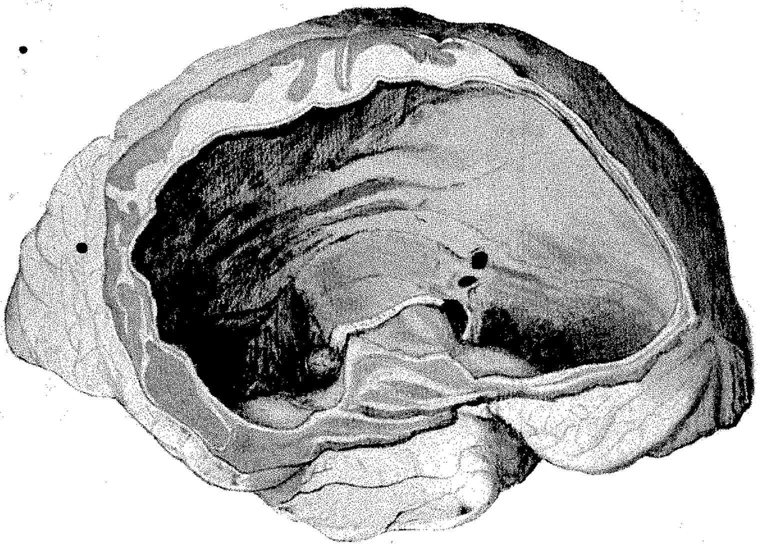
The dura did not bulge at all into the opening, and there was no pulsation, but the child's condition had rapidly failed and the absence of pulsation was thought to be connected with that fact. The flap was re-adjusted and further exploration postponed.

There had been a good deal of hæmorrhage from the flap and the bone, but not to such an extent as to raise alarm.

Before the operation ⅙ of a grain of morphia was to have been injected, but owing to a defective syringe less than that quantity was probably administered.

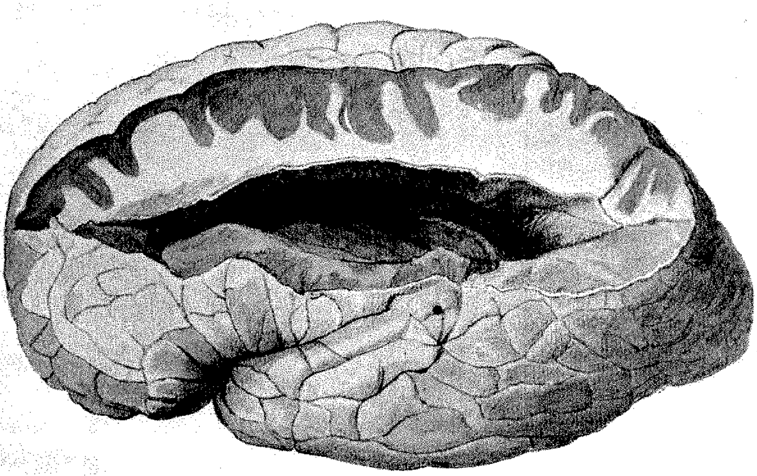
At the beginning of the operation the child's condition was good, but no chloroform was given after the removal of the second disc.

When Hey's saw was being used the anæsthetist found it necessary to inject ether, and though the operation was concluded



DRAWING OF THE RIGHT LATERAL VENTRICLE.

(Case of J. C.) [*R. C. Brown, fecit.*]



DRAWING OF THE LEFT LATERAL VENTRICLE.

(Case of J. C.) [*R. C. Brown, fecit.*]

with all despatch and means adopted to lessen shock, the pulse could not not be felt at its conclusion, and the patient's condition was very serious. He was placed in bed with the head and shoulders depressed, and for twenty minutes or half-an-hour respiration had to be kept going by slight artificial aid. Then gentle breathing became established, and it was just possible to detect the pulse. During the operation, and till death occurred, some four hours later, the pupils remained contracted and very small.

Post-mortem.—The brain only was examined. The dura mater was quite normal, and there was no opacity or thickening of the arachnoid. There was œdema of the pia mater on the convexity of the R. hemisphere over the frontal and parietal lobes. This, no doubt, was due to the operation. The surface of the R. hemisphere was fluctuant and tremulous from within an inch of the anterior extremity of the frontal lobe as far back as the occipital fissure, and laterally from half an inch from the longitudinal fissure to the sylvian fissure. The same condition was marked in the middle temporo-sphenoidal convolution. It was plain that the R. hemisphere contained a cavity full of fluid, and that its walls were very thin. Eight ounces of fluid were removed with an aspirator, and the brain was placed in a bichromate preservative solution.

The fluid was clear, of an alkaline reaction, and had a sp. gravity of 1010. It contained a trace of albumen and sugar, and a considerable quantity of salt. Microscopical examination gave no information.

Examination of the hardened brain.—The R. hemisphere was decidedly smaller than the left. This difference was not noticed at the necropsy, and was probably due to the preparation of the brain and the removal of some of the fluid contents.

In order to expose the R. ventricle, its roof was removed by a vertical incision $6\frac{1}{2}$in. long parallel to the median fissure and half an inch external to it. The extremities of this incision were joined by a horizontal cut which divided the sylvian fissure near its bifurcation. The roof so removed was $6\frac{1}{2}$in. long by 3in. wide at its broadest part.

The dimensions of the R. ventricle were :

Length	$4\frac{5}{8}$in.
Breadth in ant. cornu	$1\frac{1}{8}$in.
,, in centre of ventricle	$1\frac{1}{16}$in.
,, behind descending cornu ...	$1\frac{3}{4}$in.

Depth, everywhere about $1\frac{3}{4}$in., except at the anterior part, where it was $2\frac{1}{8}$in.

In shape it resembled the hollow cast of a large kidney, the dilated anterior cornu corresponding to the upper extremity, the posterior and part of the descending cornu, dilated into a single hollow, represented the lower extremity, whilst the concavity of the hilus was suggested by the rounded elevation formed by the corpus striatum and optic thalamus.

The *thickness* of the wall of the ventricle measured less than $\frac{3}{16}$ of an inch anteriorly, $\frac{1}{2}$ an inch posteriorly, and $\frac{14}{16}$ of an inch externally at its thickest part, where the section had traversed the temporo-sphenoidal lobe.

The anterior and posterior cornua were dilated beyond recognition, and formed part of the general kidney-shaped cavity. The descending cornu was enormously enlarged, and when the little finger was introduced to the end, its tip could be moved about as if inside the finger of a much too large thick glove.

The lining membrane of the ventricle was thick and hard, and could be partially peeled away here and there; the thickening was common to both ventricles, but the readiness with which it could be separated in the right as compared with the left was due to the firmness of the brain tissue surrounding the right ventricle.

The corpus striatum was so compressed and atrophied that its position could only be guessed at. The optic thalamus was visible in front of the choroidal plexus as an eminence $\frac{1}{2}$in. long by $\frac{3}{16}$in. wide. In front of this eminence was the foramen of Munro—an irregular opening having a diameter of $\frac{5}{16}$ of an inch. Reaching from this foramen and curving into the descending cornu was the choroidal plexus. The septum lucidum was thinned and expanded, and anteriorly two rounded apertures with smooth edges (diameter about $\frac{3}{16}$in.), and a third minute one communicated with the left ventricle. They were situated just above the foramen of Munro. The walls of the right ventricle looked, and were firm, and when the roof was removed the ventricle maintained its shape and showed no tendency to collapse. The part removed could be handled and raised by one end without any fear of damage resulting. This formed a striking contrast to the soft and succulent condition of the left hemisphere, which resembled fresh brain substance, although it had been in hardening fluids for some months.

The left ventricle was very different.—The roof was removed as on the right side, but on account of the thickness and softness of the walls it was difficult, and when it was lifted off precautions had to be taken to prevent the sides of the ventricle falling together.

The extreme length of the L. ventricle was	...	4in.
The width ,, ,, ,, about	...	1in.
The depth ,, ,, ,, about	...	½in.

These measurements cannot be considered exact, because, in consequence of the softness of the walls and the necessity for handling the parts in order to inspect them, the cavity could be made to appear either larger or smaller, but when care was taken to keep the structures as far as possible in their natural position, the cavity seemed to be approximately of the above dimensions.

The walls were nowhere less than an inch thick, and in some places as much as 1½inches.

During manipulation there was a tendency for the lining membrane with a layer of brain substance nearly ⅛in. thick, to separate from the surrounding tissue.

The anterior, posterior, and descending cornua were larger than natural, but the last-named would not admit the little finger.

The corpus striatum and optic thalamus were normal in shape and appearance; the foramen of Munro was enlarged, but smaller than that on the R. side, and had a more regular outline. The L. ventricle appeared uniformly and moderately enlarged; but there was no obvious atrophy of the surrounding structures.

Base of the brain.—On looking at the base the difference in size of the two hemispheres was very marked. The measurements were—

R. 6in. × 2½in.
L. 7in. × 3in.

The floor of the third ventricle formed a prominence as big as half a small walnut, and was about the thickness of a grape skin. It was divided by a median incision.

The third ventricle was dilated, and admitted the first phalanx of the index finger without risk of doing any damage. The R. wall was markedly concave—the left was flat.

Section through the pons showed the iter was not dilated. The tissue around it was slightly softened (*post-mortem* change?). There was no dilatation of, or atrophy of the structures in the *fourth ventricle,* and the cerebellum was unaltered.

The medulla showed descending degeneration in the usual places.

We are indebted to Professor Jacob for the following description of two microscopical sections cut from corresponding positions in the R. and L. frontal lobes.

Right.—"Considerably thinned. Microscopically has lost all trace of nerve structure, showing no nerve cells, but merely condensed neuroglia infiltrated with inflammatory products. Numerous inflammatory foci are present, and the perivascular spaces are enlarged.

"Pia mater shows marked inflammatory thickening."

Left.—"The ordinary layers of the cortex can be recognised, but the nerve cells show considerable degenerative changes, and there is much dilatation of the perivascular spaces. There is no thickening of the pia mater."

There were peculiarities in the clinical history of this case, which all who saw it during life were unable to explain upon any supposition. The pathological conditions throw light upon those clinical difficulties, and in the following explanation we shall try to show how the clinical facts and the *post-mortem* appearances can be interpreted.

The first injury occurred five years ago and, after a considerable interval of consciousness, was followed by slight signs of compression which soon disappeared. That some serious lesion had resulted was clear, for a month later a fit occurred beginning in the L. arm, and this was repeated every second month.

Now this is hardly like a history of hæmorrhage, and nothing was found at the necropsy to give colour to such a suggestion. But it is not at all improbable, in the light of subsequent events, that there may have occurred at this time a transient congestion of part of the encephalon with effusion, capable of producing slight and temporary symptoms of compression; and a bruised brain, which we might expect to be the initial factor in such a traumatic congestion, would easily explain the subsequent development of epileptiform attacks.

After an interval of two years another fall occurred. There is nothing to show whether this was the result of a fit or of accident. Concussion resulted, and from the prolonged unconsciousness (about a fortnight) that ensued it is practically certain that the patient was again suffering from compression. During a very slow recovery he exhibited for a time symptoms of cerebral irritation, and signs of spastic paralysis of the L. arm and leg gradually appeared.

He was unable to leave his bed for eight months, and the medical man who saw the patient at this time has described him as having a "purely vegetative existence."

The pathological appearances suggest that there occurred during this period:

(1) A sub-acute encephalitis involving the whole or greater part of the R. hemisphere, and producing a softening of the affected area.

(2) Inflammatory effusion into the brain substance, into the ventricles, and into the lymph spaces on the surface of the brain, giving rise to increased intra-cranial pressure and to signs of compression.

(3) Dilatation of the R. ventricle with absorption of the softened and inflamed brain tissue surrounding it in consequence of the increased intra-ventricular pressure; and

(4). From the same cause, dilatation of the L. ventricle, but without absorption, because the L. hemisphere was not involved in the inflammation, and was of normal consistency.

(5) Loss of function of those parts of the brain which had been or were inflamed.

Prolonged increased pressure would lead to absorption and recession of the softened inflamed brain tissue wherever it was exposed to it, whether upon the surface of the brain or within the ventricles, and since the ventricles are in direct communication with the lymph spaces upon the surface of the brain, any increase of pressure within the ventricles would speedily equalize itself throughout the whole cerebro-spinal canal.

In order, therefore, to explain why absorption of brain tissue has occurred only at the expense of the softened parts of the ventricle walls *from within*, it is necessary to account for the intra-ventricular contents being maintained at a higher tension than normal, whilst the pressure upon the surface of the brain hardly varied from what was natural; and this can be done if a plausible reason can be suggested to show that the intra-ventricular fluid was prevented escaping by the iter.

The maintenance of an ordinary pressure upon the surface of the brain is easily understood, for any temporary increase of fluid resulting from inflammatory effusion would speedily be drained away into the general circulation.

Now when the R. hemisphere was inflamed the adjacent and continuous structures could hardly have been so fortunate as to escape entirely without feeling some effect from the mischief, even supposing that they were not directly involved; and it is not difficult to understand how so small a channel as the iter might be occluded by the pressure of inflammatory effusion or œdema in its surrounding tissue. And a further possibility may be mentioned—the orifice of the iter in the third ventricle may have been obstructed by an œdematous ependyma. In one or both of these ways in all probability the excess of fluid secreted into the ventricles was prevented escaping, the intra-ventricular pressure was raised, dilatation of both lateral and the third ventricles took place, and on the R. side the dilatation was not only more marked because this hemisphere was inflamed and soft, but was materially assisted by absorption from pressure of the damaged brain tissue, and at a later period doubtless by the contraction of the organized products of inflammation. When the encephalitis subsided the effusion into the cerebral tissues would be absorbed, the iter once more become patent, and the normal equilibrium of the cerbro-spinal fluid be restored.

If we consider how much of the substance of the R. hemisphere must have been destroyed at this period, partly from pressure and partly from the contraction of cicatricial tissue resulting from inflammatory exudation, there is no difficulty in accounting for the gradual onset of spastic paralysis in the L. arm and leg, which developed during the eight months the patient was confined to bed.

But what is the meaning of the peculiar condition of apathy in which the patient lay during the eight months following the period of compression?

It is probable that after the first two months the brain was in very much the same state as that in which it was found at the *post-mortem*, and it may be that the cause of this condition is to be sought in the loss or diminution of function that most structures are subject to during and after inflammation. This would explain not only the tedious and very gradual recovery, but also the recurrence of fits which took place soon after he was sufficiently improved to be able to get out of bed, and which had been in abeyance from the time he met with his second fall. As the period of impaired functional activity gradually passed away the power to respond to irritation would reappear in those ganglionic and conducting structures that had not been too seriously damaged to recover.

Lastly, it must not be forgotten that at the time of the operation, and no doubt for months and even years before, the condition of parts was stationary so far as the macroscopic lesions were concerned;—one ventricle was enormously and the other moderately dilated; both were filled with normal cerebro-spinal fluid at the ordinary tension and free from all inflammatory elements. The conditions present were the result of a long past inflammation. The cerebro-spinal fluid was fulfilling its function of properly supplying the vacuum within the cerebro-spinal canal, and it was present in such large quantities because the potential vacuum had been so greatly increased.[1]

In attempting to discuss diffuse inflammation of the brain it is very difficult to find firm ground from which to make a start.

The microscopic appearances of chronic inflammation and sclerosis are apparently indistinguishable, and in doubtful cases the decision between inflammatory or purely degenerative changes can hardly fail to be arbitrary; and to those who are not experts in the pathology of the nervous system the indifferent application of terms, implying different processes of disease, to a particular morbid condition, is very puzzling.

The obscurity surrounding diffuse encephalitis is apparent in the account of inflammation of the brain given by Mr. Jonathan Hutchinson in "Quain's Dictionary of Medicine," vol. i., p. 148 (1883).

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[1] The authors trust that the full description they have given of a case to which they have been unable to find a parallel may not be thought unnecessary. Full of interest to the student of medicine, it is most instructive as an illustration of one of the pitfalls that may be encountered in Cerebral Surgery.

"It may, perhaps, be doubted whether the occurrence of diffuse inflammation of the brain substance as an acute disease has as yet been proved, excepting as a result of wounds. Even as a traumatic lesion, its special features have by no means been accurately studied. It is, however, highly probable that after penetrating wounds of the brain, its substance may inflame, just as the cellular tissue of a limb may, the inflammatory processes beginning at the site of the wound, and rapidly spreading through a large part of the hemisphere. It is probably in the perivascular spaces that the process chiefly spreads, and it is in these that the microscope will detect the most abundant results. Such a condition of diffuse encephalitis may exist without there being any visible changes in the brain. It may, perhaps, be a little softened or a little congested, but very probably there is nothing about which the most experienced pathologist could feel certain until the microscope is resorted to."

"It is not possible, in the present state of our knowledge, to speak with any certainty of the symptoms of diffuse encephalitis. They will vary, of course, with the region affected; and disturbance of function, followed by, more or less, complete loss, will be the most frequent occurrences."

In the hope of learning something more definite about diffuse inflammation of the brain, we have searched through a good deal of English literature, and have collected the following cases:—

Case 1.—*Wilks and Moxon* mention, as an example of acute general cerebritis, the case of a young man, who was admitted into hospital supposed to be suffering from fever on account of the torpid state into which he had fallen; he never spoke, but lay perfectly motionless in bed, with his eyes open, apparently watching those around him. His illness lasted a few weeks; and after death the whole of his brain was found soft and pulpy, whilst some parts were semi-fluid.—"Pathological Anatomy," p. 237.

Case 2.—A man of 40, after frequent pain and throbbing in the L. side of the head, had convulsive paroxysms in the R. arm and leg, which were followed by giddiness, confusion of thought, and torpor of the R. side. Choreic movements were next succeeded by paralysis: speech gradually was lost, and also power over the sphincters; but the mental faculties seemed unaffected. He often screamed, as if in pain; and, after seven months' illness he became comatose, and died in three days.

"The L. hemisphere was soft and fluctuating throughout its whole extent, like a bag of fluid. On cutting into it there was about half-an-inch in thickness of sound cerebral substance; the remainder of the hemisphere was found nearly reduced to a fluid mass, partly consisting of purulent matter, and partly of cerebral substance in a soft pulpy state, but the greater part was purulent. From the mass of disease the ventricle was separated merely by the membrane which lines it, and contained a small quantity of serous fluid." There was a clot, the size of a walnut, in the left thalamus.—"Pathological and Practical Researches on Diseases of the Brain and Spinal Cord." 3rd edition. Abercrombie. Case 38, p. 94.

Case 3.—A complicated case. A middle-aged man one day had a fit for the first time in his life. The next day he fell in the street, and was picked up insensible, with a scalp wound. On rallying from a lengthened collapse he was very violent. For some days he suffered from intense pain in the head. On the seventh he became comatose and was trephined in consequence of an unfavourable appearance of the wound. He died the same night. *P.M.*—There was some serous effusion between the arachnoid and pia mater, especially at the base; the membranes were quite transparent; the ventricles were dilated and filled with a large quantity of fluid and the brain itself was watery throughout and soft. There was a small tubercular deposit as big as a pea, surrounded by a small area of softening and injection in one of the basal convolutions.—"Holmes' System of Surgery," vol. i., p. 623.

Case 4.—A boy, aged 13, died three weeks after he had been thrown from a donkey and fallen on the back of his head. The ventricles were enormously dilated and filled with serum and recently effused lymph, and the central white parts of the brain were very soft and broke down when slightly touched. The grey and white substance of the brain were throughout very much congested, and there was extensive inflammation of the membranes. There was a slight fissure of the occipital bone.—"Holmes' System," vol. i., p. 622.

Case 5.—A man, aged 37, died twenty-four hours after an injury of the head in a fall from a tree. The ventricles were found filled with fluid, and the fornix, remarkably soft, gave way when slightly touched. At the base of the brain around the pons and medulla oblongata ther was a large quantity of milky puriform fluid; the substance of the brain was firm, and the puncta of blood larger and more numerous than usual; the cribriform plate of the ethmoid was broken and the corresponding part of the brain bruised.—"Holmes' System of Surgery," vol. i., 622.

Case 6.—*Wilks and Moxon* mention the case of a girl supposed to be hysterical. She was perfectly helpless, could scarcely move her arms, spoke and swallowed with difficulty, and had a stupid expression. After death the pia mater was found thickened, of a yellowish colour, and adherent to the brain, especially over the frontal lobes. There was found a general hardening of the brain, especially of the anterior lobes, which could be grasped like a hard tumour. The microscope showed the nerve fibres had almost disappeared, and their place was occupied with granule masses, vessels and fibrillated tissue.—"Pathological Anatomy," p. 238.

Case 7.—A girl of 14, in so helpless a condition of mind and body as to be at first mistaken for an idiot, had been an intelligent child a year before her death. She lay quiet in bed in a state of vacuity. At the *post-mortem* it was found she had been the subject of general meningo-cerebritis; the arachnoid was thick and opaque, and the pia adherent. The brain as a whole was much firmer than natural, and felt hard to the touch. No cause for this condition could be found.—Wilks and Moxon, "Pathol. Anat.," p. 238.

Case 8.—Dr. Bright in his medical reports relates the case of a little girl, who, for a year before her death, lay in a perfectly motionless and senseless state, with her limbs stiffly extended, and without the possibility of making the slightest movement. After death the white substance of the brain was found almost as hard as cartilage, and a stream of water washed off the grey matter, giving it the appearance of a wax model of the brain. The ventricles also looked as if they had been modelled in wax.—Wilks and Moxon, "Path. Anat." p. 238.

Case 9.—Dr. Henry Ashby records the case of a boy who died at the age of 20 months. Three days after birth he began with fits; when eight months old he was apparently an idiot, and blind and deaf, and the R. arm and leg were flexed and more or less stiff. The rigidity increased; the symptoms of descending degeneration were more marked on the right side than on the left. He lay in bed with head retracted and face turned to the L. side. At the necropsy the arachnoid was thickened and opaque, and the pia consisted of many tortuous vessels. There was no optic neuritis or atrophy. The brain substance was remarkably hard and firm, and apparently shrunken. That between the cortical grey matter and the basal ganglia was firm and dark in colour, and evidently sclerosed. The lateral ventricles were much dilated. There were descending changes in the cord and medulla. The microscope showed the neuroglia to be exceedingly coarse and granular, the perivascular spaces dilated, and in places an increased amount of fibre tissue accompanying the vessels in their course.

Dr. Ashby says the most plausible explanation of this condition of sclerosis is that it is due to meningo-encephalitis of the convexity, which took place during fœtal life, and which was followed by atrophy of the convolutions and descending sclerosis.—*Pathological Society's Transactions*, vol. xxxvii., p. 12.[1]

These nine cases present us with two distinct forms of encephalitis. The chief pathological feature in the one is *softening*, and in the *other hardening* of the brain tissue; but in all the changes are *diffuse.*

In the cases in which there was softening (1 to 5), and in which the inflammation was more or less acute, the changes involved either the whole brain, or one hemisphere, or central softening was accompanied by increased vascularity of the remainder.

It is, further, to be noted that in one of the cases where the latter condition was found, 24 hours only had elapsed between the injury and death, and the membranes at the base were presumably affected. In the other, after three weeks, in addition to a very marked congestion of the unsoftened parts, there was extensive inflammation of the membranes.

The *duration* of the disease varied from 24 hours to a a few weeks, but in one case, with peculiar features, it was seven months.

Traumatism was the exciting cause of the inflammation in three cases (3, 4, 5,) but in only one (5), was it probable that the membranes had been penetrated, and in that the softening was found in the central parts; and in another (3) it is uncertain what predisposing effect a small previously existing tubercular focus may have exerted.

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[1] Two cases are recorded in "Fagge's Medicine," p. 554, vol. i. One is probably a fuller account of Case 7. The other is the case of an infant who had an illness of 14 months, marked by convulsions, followed by long periods of unconsciousness. The hemispheres were indurated and the pia adherent.

In the other two cases there is nothing to suggest the cause of the disease.

In case 2, though the pathological appearances were those of a diffuse encephalitis the clinical history suggests that the disease took a course very different to what occurred in the others. The way in which additional symptoms slowly made their appearance, impairment of function preceding its abolition, point to a gradual implication of portion after portion of brain until the whole hemisphere was converted into a semi-purulent mass. Such a condition from the chronicity of its course, and the formation of pus presents some likeness to a cold abscess, and it serves to illustrate the difficulty of deciding in some instances to which category, acute or chronic, localised or diffuse, a particular case is to be relegated.

In cases 6, 7, 8, 9, *there was a general hardening* of the whole brain; and in three of them, at least, the membranes had been the subject of a chronic inflammation.

The disease in all was probably identical, and the hardening due to an increase in the fibrillated tissue throughout the organ and along the course of the vessels.

In the three cases in which the *duration* is stated or can be inferred, the disease lasted from 12 to 20 months.

In no case is there any history of traumatism, and in all the cause was obscure.

The patients were all young, probably all under 20.

In Ashby's case the disease originated as an intra-uterine condition, and this is the only instance in which the records, we have had access to, note the occurrence of convulsions.

In the only case in which the state of the optic discs is mentioned it was normal, though the infant was thought to be blind.

The most prominent symptom, and one which was present in all these cases, was the *state of vacuity and helplessness* in which the patients lay for long periods. The impairment and abolition of function was evidenced in the loss of intelligence and muscular power more or less complete. We believe that *this long continued torpor of mind and body* is the most salient feature of chronic diffuse encephalitis.

Allusion has already been made to the fact that in the chronic cases with hardening, the whole brain was affected. In the acute form, if the patient survives long enough, the same torpor may be present when the whole brain is involved—(case 1); but in acute cases of short duration, this valuable symptom may never develop, as signs of concussion or compression are likely to predominate from the first.

Therefore, though this condition (torpor of mind and body) may be present in acute diffuse encephalitis, it is more especially characteristic of the chronic form in which induration is the chief pathological feature, and it is, then, usually of long duration.

The symptoms due to secondary degeneration in the cord call for no remark.

THE PHYSICS OF THE CRANIAL CIRCULATION AND THE PATHOLOGY OF HEADACHE, EPILEPSY, AND MENTAL DEPRESSION.

BY A. HAIG, M.A., M.D.OXON., F.R.C.P.

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In the Spring number of BRAIN, 1891, I pointed out that certain cases of headache, epilepsy and mental depression bore what appeared to me to be an important and fairly constant relation to the excretion of uric acid in the urine.

I suggested that an excess of uric acid relatively to the urea in the urine was an index of an excess of urate in the blood, and that such excess of urate, by contracting the arterioles and capillaries might produce cold skin and extremities and scanty urine, and by effecting similar changes in the intracranial circulation might produce the headache, the fits, or the mental depression.

Further than this, I did not at that time attempt to go: but I believe that it is now possible not only to bring forward further proof that urates have the action on the vessels which I then attributed to them; but that this vascular effect of urates being granted it is now no very difficult matter to show by the aid of the researches of others that the changes in the intracranial circulation which urates may thus bring about may be quite sufficient to produce headache, fits, or mental depression.

The subject, therefore, naturally divides itself into two parts. (1) The proofs that urates do contract the arterioles and produce high arterial tension. (2) The effects of such high arterial tension on the intracranial circulation, and to these I shall add: (3) A short review of the symptoms and pathology of these disturbances regarded from this point of view.

As regards the effects of urates on the circulation I have previously mentioned that in migraine the pulse is well known to be slow and of high tension, while the urine is scanty and the skin and extremities cold, and some six or seven years ago I pointed out that the urine excreted during an attack of severe headache contains a more or less large excess of uric acid relative to the urea excited at the same time, and I suggested that the excess in the urine was the index of a relative excess of urate in the blood.

I further pointed out that by attending to certain laws that appeared to govern the solubility of urates it was possible to increase or diminish at pleasure the excretion of urates in the urine, and that along with such increased excretion, however produced, went high tension and slow pulse, cold skin and extremities, scanty urine, mental lethargy and depression, or headache; and along with diminished excretion, however brought about, there went quick and relatively low tension pulse, profuse urine, warm skin and extremities, mental brightness and well being; so that it is possible, as I have said, to influence the pulse rate and tension, and the mental condition to almost any required extent, and did time and space permit I could now bring forward scores of cases in which I have done this, and not a few in which others have been able to do the same.

See among others Dr. J. T. Eskridge in the *Alienist and Neurologist*, Jan., 1892, for some interesting cases of headache, depression, and hysteria, where drugs and other treatment acted just in the way that I have described.

But there is a further fact which now testifies to the truth of my reasoning, for not only is the urine scanty in the uric acid headache, and profuse when it passes off, and the same in epilepsy and hysteria, that is to say, the excretion of water varies in these diseases inversely as the uric acid, when the urate excretion is high the water is low, and *vice versa*; but the very same thing holds in physiology also; so that in every one and at all times the urinary water is inversely as the urate excreted along with it.

If this is a fact (a physiological fact) it is alone sufficient to prove the influence of urates on the vessels, for what other possible explanation can be given of the constant relation of the excretion of urates to the excretion of water.

Further, in a recent paper in the *Lancet* (July 9th, 1892), "On the elimination of water and the pathology of dropsy," I was able to show that this law of the inverse relation of urates to water dominated and explained a whole host of well-known facts in therapeutics and pathology.

That in therapeutics all drugs which clear urates out of the blood and diminish their excretion in the urine do at the same time relax the arterioles, and produce a diuresis, and that the well known diuretic action of such drugs as mercury, opium, iodide of potassium, and other iodides, the salts of lithium, and the salicylates, etc., is a simple illustration of this law, which completely explains their action.

And conversely that drugs which increase the urate in the blood and urine, such as the alkaline salts of sodium, and potassium, the salicylates and the neutral phosphates, do, while the increased excretion of urates lasts, produce contracted arterioles, slow high tension pulse and scanty urine.

I also pointed out that in pathology the same law holds, that in all diseased conditions which tend to bring an excess of urate into the circulation or retain it there, we have contracted arterioles, slow high tension pulse, and scanty urine. Among such conditions I have specially mentioned migraine, some cases of epilepsy, certain menstrual troubles, especially dysmenorrhœa, many conditions of low health and nutrition, as the failure of nutrition in old age, and after disease; Bright's disease, both acute and chronic, Raynand's disease and paroxysmal hamoglobin-uria (see *Transactions of the Medical Society of London*, vol. xv., and *St. Bartholomew's Hospital Reports*, vol. xxviii.). In all these conditions we have slow high tension pulse and scanty urine, and according to the way in which the high tension affects the intracranial circulation in a given case, we have head-ache, mental depression or fits (epileptic or hysterical), and other manifestations of epilepsy or hysteria, not to mention syncope, cerebral hæmorrhage, and a host of other structural and nutritional alterations.

Conversely the pathological condition (fever) which most quickly clears the blood of urates and diminishes their excretion in the urine (driving it, as we often know to our cost, into the fibrous tissues, the joints, liver, spleen, &c.) does at the same time relax the arterioles, producing quick low tension pulse, and at its first outset a more or less marked diuresis.

So much is this the case that I offered (*Lancet*, prev. ref.) to tell the days in any chronic hospital case on which the temperature had risen above normal simply from the record of the urine passed, if no drugs affecting the problem were being given.

I assert, therefore, without fear of contradiction, that in physiology, pathology, and experimental work in man, the urinary water is from hour to hour, or from day to day, inversely as the uric acid excreted along with it; and that drugs which increase the excretion of urate diminish the water and *vice versa.*

The pulse rate and tension bear also precisely the same relation to the urate excretion, and they do this because the urate excretion is to a large extent an index of the amount of urate in the blood (Bright's disease being an exception to this rule for reasons given elsewhere; see "Uric Acid," J. and A. Churchill, 1892, p. 225.)

The one series of facts explains the other; the water is scanty because the arterioles are contracted, and I have pointed out that Dr. Lauder Brunton has shown that drugs like digitalis, which contract the arterioles, may diminish or even completely stop the flow of urine (see "Uric Acid," p. 100.)

The heart's action is slowed because the contracted arterioles raise the tension, for Marey has formulated the law that the pulse rate is inversely as the arterial tension.

There is an exception to this law which is also frequently met with in Bright's disease, for contracted arterioles and high tension in this disease are not always accompanied by a slow pulse, and the explanation of this is, I think, as follows:—Marey's law, I believe, holds good so long as the heart is up to its work, so long as its systoles are complete, but if the heart begins to give way, and the ventricle dilates, its systoles are imperfect and must be repeated more often to keep up the circulation.

Thus, in acute Bright's disease with heart failure, or at the end of a chronic case as dropsy comes on, you find in the heart increased area of dulness, apex to the left, 1st sound reduplicated, and 2nd sound loud, but the pulse instead of being slow is 85 to 90 in the minute; here we have cardiac failure with dilatation.

And there is this marked difference between the slow and the quick pulse in Bright's disease, as regards the effect of drugs.

Take a case of Bright's disease in which the heart is strong and compensation is perfect, there are all the cardiac and vascular signs of high tension, and the pulse is slow, say 45 to 55. Give any of the drugs which clear urates out of the blood, acids, the nitrites (which act as acids, see "Uric Acid," pp. 40-41), opium, mercury, or iodides, you relax the arterioles, increase the urine, and generally (in my experience) diminish the absolute quantity of albumen, and the pulse will quicken up to 70 or 80.

But take a case of Bright's disease, in which owing to cardiac failure, the pulse is quick; give any of the above drugs as before to relax the arterioles; you will increase the urine and diminish the albumen as before, but as you do this the pulse will get slower, instead of quicker. As you relieve the heart from the load of high tension against which it had to work, its systoles become more complete, and it alters its rate of action in the direction of the normal, that is, the pulse becomes slower instead of, as in the other case, faster.

With regard to my statement as to the inverse relation of urates and urinary water there are a few things which might appear to anyone taking up the subject *de novo*, to be exceptions, when they are not so in reality.

Thus it is obvious that relaxing the arterioles cannot cause diuresis when there is no available water in the body, and conversely when, as in dropsy there is a great excess of water in the body, relaxing the arterioles will cause great and lasting diuresis, which continues till the dropsy is all gone (see interesting case mentioned in "Uric Acid," p. 225).

But to return to the other condition, there may be no available water because profuse perspiration, diarrhœa, or previous diuresis has removed it all.

Thus a diuresis, at any given time, is generally due to two factors: 1st, that the urates have been cleared out of the blood, and the arterioles relaxed as a consequence of this, and 2nd, that some previous contraction of arterioles has kept back and retained in the body some water which now has an opportunity of passing out.

I have often previously mentioned the fact that in physiological conditions the water is least in the day, say 40-50cc. per hour, and most in the night, as 70 to 80cc. per hour, this being just the reverse of the urate excretion; but the diuresis in the night is the result of the retention of water during the day, and is generally proportioned to it: *i.e.*, if there is a day of more than usual high tension with it results headache, mental depression and scanty urine, and if at night the arterioles are relaxed and the tension falls, the urine will be more than ordinarily profuse, being double the hourly excretion of the day, and far more per hour than on any night corresponding to a day of ordinary tension.

In Bright's disease this is nearly always the case, and the urine of the 12 hours ending 8.0 a.m. is very often double that of the 12 hours ending 8.0 p.m. In other words, the urine in chronic Bright's disease is only profuse at night; by day the tension is high and the urine scanty.

Further, by altering the time of the urate excretion, one can alter that of the water excretion also; thus let anyone take such a dose of alkali (not lithia, for reasons given in "Uric Acid," p. 29) as shall render his urine alkaline all through the 24 hours, and continue this for seven to eight days. After two or three days, he will find that the urine is now much the same in quantity per hour both night and day, that it is not scanty during the day and morning hours, and therefore not profuse at night.

And the reason of this is obvious: the plus excretion of urate which normally takes place in the "alkaline tide" of the day is the result of the retention of urate during the acid tide of the previous night, but he has been taking alkali for several days, and there is now no acid tide of the night, and its effects have disappeared with it; there is now little or no retention of urate at night, hence there is no plus excretion of urate in the alkaline tide next day. As there is no plus excretion of urate by day, its effects on the arterioles and the excretion of water are absent; there is no excess of it in the blood, no holding back of water, and, consequently, no diuresis the following night.

Indeed, under these circumstances, the urine may be quite profuse by day, and then it is scanty at night, because all the available water has been run off by day; this being just the reverse of what takes place when the acidity is normal.

This is only one instance of the way in which, by controlling the urate, we can control the water, and, at the same time, alter also at pleasure the rate of the heart's action and the tension of the pulse.

From these and many other facts which there is no space to give in full here, I infer and assert without fear that, other things being equal, the contraction of the peripheral arterioles throughout the body is directly proportional to the amount of urate that is circulating in the blood.

I do not assert that there are no other substances which produce the same effect, but I believe that in the human body urates are more important than any other substances or combination of substances, and this is the reason of the position which I have found them holding in relation to certain disease processes about which I have written.

Let it be granted, then, that contraction of the arterioles is proportional to the urate in circulation; we can now pass on to our second problem, viz., how does the high tension produced by this contraction of arterioles affect the intra-cranial circulation, and how may these effects help us to elucidate the pathology of those cerebral disturbances, which I have found by observation to bear a special relation in time to the excretion of uric acid?

While thinking over this problem, I became greatly interested in some articles on the intra-cranial circulation, which appeared in "Virchow's Archives," by Dr. Geigel (*Archives*, vol. cxix.), Prof. Lewy (*Archives*, vol. cxxii.), and an account of an experimental investigation by Prof. Dr. H. Grashey ("Fest Schrift München," 1892).

I shall mention these here, however, only so far as they appear to me to bear on our problem.

Geigel's main contention is that in the conditions which obtain within the skull dilatation of an artery, does not increase the flow of blood through the brain, but really diminishes it, because it compresses the veins and capillaries.

Lewy dissents from this in so far as it refers to physiological conditions, as he points out that in these conditions the lymph channels and capillaries are so numerous that they will bear a good deal of compression before they really diminish the blood flow through them, but he acknowledges that in disease, when part of the available space has been already taken up, say, by a cerebral tumour, or where some of the available capillaries have been blocked or destroyed by meningitis, it might then be, as Geigel asserts, that dilatation of an artery might actually diminish the quantity of blood flowing through the brain.

Both these writers agree with each other and with a previous writer (Althann), whom they quote, that it is not important how much blood is present in the skull; but how much flows through the brain in a given time, because on this depends that satisfactory interchange between the blood and the tissues on which the functions of the brain are so absolutely dependent.

Grashey appears to believe in this also, but his investigations are in great part directed to the determination of the effects on the intra-cranial circulation of increased arterial pressure, and bear therefore in the most direct manner upon our problem.

He tells us (previous reference, p. 73) that as regards the nourishment of the brain, it is, apart from the quality of the arterial blood, of more importance how much streams through the cerebral vessels in the given time.

He then points out that his researches show that, other things being equal, the quantity of blood which streams through the cerebrum in a given time rises with the pressure in the aorta up to a certain point, but when that point is overpassed the intra-cranial pressure is so great that the central veins are compressed and begin to vibrate, and with this vibration there is always a considerable reduction in the amount of blood passing through ; and though the veins and capillaries behind the point of vibration are overfilled with blood, the conditions become unfavourable to the nutrition of the brain.

But, he continues, since the cerebro-spinal fluid comes from the blood, it is not improbable that the stasis in the veins and capillaries thus brought about may result in an increase of its quantity, and so we get a further increase of intra-cranial pressure, so that when once the pressure rises so that the central veins begin to vibrate, a vicious circle comes into existence, which must bring danger to the brain.

We shall see, I think, further on that this suggestion of Grashey's with regard to the effects of high pressure and stasis on the quantity of the cerebro-spinal fluid may help us to explain some points in the pathology of uræmia.

He thinks that the veins are specially compressed at their point of entry into the cerebral sinuses, and he does not believe that high pressure compresses the capillaries ; on the contrary, they are overfilled with blood, owing to back pressure from the veins.

We have, then, as the result of these researches and my own, certain facts as observed, and what seems to me a very simple explanation of their inter-relation and causation.

The facts are those observed by myself, and mentioned in my previous paper and elsewhere, namely, that certain forms of headache, epilepsy, and mentral depression bear an important relation in time to the excretion of urates in the urine, and that by controlling the formation and excretion of urates, these disturbances can also, to a large extent, be controlled.

I have explained these facts by suggesting that the excessive excretion of urates in the urine is the sign of a similar excess in the blood, and that when we alter the amount in the urine we at the same time alter the amount in the blood ; and have suggested that the relation of these disturbances to each other and to the excretion of urates was due to the fact that excess of urates in the blood affected the intra-cranial circulation, and so altered the functions of the brain.

It appears now from the interesting researches of Professor Grashey that we can go a step further and point out the exact way in which urates act on the intra-cranial circulation, namely, by producing high arterial tension.

The effects of uric-acidæmia on the skin and extremities, causing them to be cold and pale, is easily explained by the contraction of arterioles and capillaries which it produces in these parts. The same effect on the arterioles of the kidney may also suffice to explain the scanty urine of uric acidæmia. But we are met by a difficulty when we come to apply the same reasoning to the intra-cranial circulation, for it has been pointed out by Professor Horsley ("Origin and Seat of Epileptic Disturbance," *British Medical Journal*, vol. i., 1892, p. 63), that his experiments all go to show that the condition of the cortex during the epileptic attack is one of congestion, not of anæmia.

It appears then that if the uric acidæmia is the cause of headache, epilepsy, and mental depression, that it must act on the brain in a somewhat different manner from that in which it acts on the skin and kidneys.

And Professor Grashey's researches suggest at once an explanation of the difficulty.

Uric acid, when it contracts the arterioles of the skin, kidneys, and other organs, not only affects these organs, but produces a general effect throughout the whole body, namely, high arterial tension; this high arterial tension will, doubtless, affect the intra-cranial circulation in the manner that Grashey describes, and, producing more or less stasis there, will quite account for the headache, epilepsy, and mental depression, and their time relation to an excessive excretion of urates.

This also explains completely Professor Horsley's observation, for the intra-cranial condition is one of stasis and hyperæmia, not of anæmia, and the stasis and hyperæmia are due to the compression of the veins by the high pressure in the arterial system, so that we have high pressure between the tense arteries behind and the compressed veins in front, and with this stasis and deficient interchange between the blood and the tissues.

Our chain of causation will then run as follows:—(1) Uric acidæmia due to very numerous causes mentioned elsewhere, which are, however, to a large extent, under our control; (2) general contraction of arterioles and capillaries producing high arterial tension; (3) the results of the high tension, more or less stasis and hyperæmia in the intra-cranial circulation, producing in accordance with the various anatomical and physiological conditions present in a given case, headache, mental depression, or fits.

It will be found, I think, that this completely explains the observed relation in time between the excretion of urates and these functional disturbances, and will also enable us to explain more completely than has previously been possible a whole series of points in their causation and relation to other diseases.

The relation of migraine, or as I have called it the uric acid headache, to uric acid is as follows:

It is contemporaneous with a large excretion of uric acid in the urine having a relation to Urea of 1-18 or 1-20, the absolute hourly excretion is also large, say .6-.7 gr. per hour = to 14-17 grs. in the 24 hours, in a person whose normal excretion in the 24 hours would be from 10-12 grs. When a headache has gone, or has been driven off by drugs, it will be found that the hourly excretion has fallen to about .4grs. per hour.

Again, the plus excretion which accompanies the headache is preceded by a minus excretion, and if a line be drawn to represent the normal relation of uric acid to Urea in excretion at 1 to 33, it will be found that the excursion below that line which precedes the headache is almost exactly equal to the excursion above the line which accompanies the headache.

In a word there is a fluctuation in the excretion of urates, a minus excretion preceding the headache, and a corresponding plus excretion accompanying it.

Now it is quite impossible to prevent these fluctuations in the excretion of urates; they occur in everyone, and almost every hour of the day, as the result of a whole host of causes which it is impossible to shut out; is it then impossible to prevent these headaches?

This does not necessarily follow, for the headache does not depend on the fact that there is a fluctuation in the excretion of urates, but only on the size of that fluctuation.

Supposing, as I have done for reasons given elsewhere (see "Uric Acid," p. 12) that all the uric acid which is in excess of the relation to Urea of 1 to 33 passes through the blood, it is obvious that an excretion of 1 to 20, on an average urea formation (and it may be considered that the urea excretion generally equals its formation, because it is soluble) of 600 grs. will furnish 12 grs. of uric acid to pass through the blood; while an excretion of 1-20 on a urea formation of 300 grs. would only furnish 6 grs. of uric acid to pass through the blood.

By reducing our whole nitrogeneous metabolism it is thus obviously possible to prevent any large amount of urate from ever, in physiological conditions, finding its way into the blood, and if the headache is due to the absolute quantity of urate in the blood it must thereby be prevented.

Now this is exactly what I have done. I have reduced my average urea formation from 500 grs. or more which it used to be when I took ordinary diet, to 350 grs. a day now that milk and fish in small quantities form the only animal food.

Contemporaneously with this change my headaches fell in frequency and severity, and in 1891, I had only 5, and in nine months of this year only 3; while on ordinary diet they used to range as high as 40-50 in a year.

And that this change is due to the change of diet is evidenced, not only by the fact that the two changes were contemporaneous, but by the further fact that if I alter my diet and increase my urea I promptly begin to suffer again, and, indeed, I believe that but for occasional and unavoidable indulgence in meat, I should now never have a headache at all.

And there is one further fact, namely, that every single drug (and there are at least a dozen of them that are well-known) that diminishes the excretion of urates in the urine, at the same time relieves or cures the headache, and the headache is proportional, not to the amount of the drug given; but to the effect of that drug on the urate excretion.

It is, therefore, never safe to say this drug is no use. I have given so many grains of it, and the headache continues; examine the urine and you will see that the large excretion of urate continues also; either your drug has not been absorbed owing to a sick stomach, or its effect on the solubility of urate has been counteracted by some other condition present, as for instance a large amount of alkaline phosphate or other alkaline salt in the blood.

Such is the relation of this headache to uric acid, and it has long appeared to me that the only possible explanation of these facts is the one I have given, that the excess of uric acid in the blood (uric-acidæmia) produces the headache.

I think we can now see pretty clearly the way in which it effects this result.

I will also say shortly that a very considerable number of cases of epilepsy, hysteria, mental depression and irritability bear precisely the same relation to the excretion of urates that the headache does, and they also are similarly relieved or cured by drugs or diet that tend to clear the blood and urine of urates, or diminish its formation.

The great point in the diagnosis of uric acid disease is that it is temporary and fluctuating in accordance with the fluctuations in urate excretion, and is not permanent and continuous.

If a headache lasts day after day for a week and does not vary with the natural variations in the excretion of urates, or such artificial variations as can be produced by drugs, it is not due to uric acid, unless Bright's or other organic disease is also present, when we may have an organic and not a functional uric acidæmia.

If fits, mental depression, or irritability, are similarly without fluctuation in accordance with urate excretion, they also are probably not connected with it, and search should be made for organic disease.

I have shown above that contracted arterioles and capillaries, and the high arterial tension they produce, bear exactly the same relation to the excretion of urates and the fluctuation that can be produced in this by drugs and disease as the above functional disorders do; and the reason of this is that the high arterial tension is due to the uric acidæmia, and this in its turn is the cause of such serious disturbances of the intra-cranial circulation as may easily account for the origin of these functional disturbances of the nervous system, and even more serious troubles.

If, then, it be true, that certain cases of headache, mental depression, and 'fits', are due to the effects of high arterial tension on the intra-cranial circulation, those things which tend to raise arterial tension will do harm, and those that tend to reduce it will do good. Let us apply this rule to our knowledge and experience of the things that seem to condition these disturbances.

Now high arterial tension varies, chiefly with two factors (1) the contraction of the arterioles and capillaries, and (2) the power of the left ventricle of the heart to overcome the intra-arterial pressure at each systole.

Therefore, arterial tension can be lowered by two things, namely, anything that will relax the arterioles and capillaries, or anything that will weaken the left ventricle and prevent it discharging the whole of its contents into the aorta at each systole.

A great rise in arterial tension is produced as we know also by muscular exertion, though the effect is only temporary, and a great fall in arterial tension can be produced in a given region by compressing the main artery of that region.

Now it is a characteristic feature of the uric acid headache that it is worse on assuming a stooping posture and on making any muscular exertion or effort; but if it is due to anæmia of the brain it is not very easy to see how either of those acts can increase the anæmia. On the other hand, if it is due to high arterial tension compressing the veins, it is clear that muscular exertion which raises the tension will do harm, while the stooping posture will facilitate the inrush of arterial blood, but still further impede the outflow of the venous blood.

Further, it is obvious that pressure on one or both arteries in the neck will reduce the arterial tension in the skull, and so relieve the pressure and pain, which in my experience, it at once does, while if the disturbance were due to anæmia it must obviously increase it.

But muscular exertion has another effect if it is persevered in, for it produces relaxation of arterioles and a fall of arterial tension, and I have been told by numerous sufferers (and have myself also often tested the same point) that though exertion at first makes the headache much worse it eventually cures it if persevered with. Let any sufferer put on his hat and go for a sharp walk; his headache will be much worse at first, but at the end of say half an hour, he will have got thoroughly warm, his arterioles will be everywhere relaxed and his pain will have vanished, and he will once more be able to take a cheerful view of things.

This same relaxation of arterioles relieves at once both headache, mental depression and irritablity, for all are due to the same high tension, and which of them will be most marked in any given case depends to some extent on the height of the tension, but also probably on the anatomical conditions, which influence in one way or the other the effect of the high tension on the intra-cranial circulation.

Dr. Liveing tells us of the interesting results obtained by Dr. Parry in cases of this headache, also in hysteria, epilepsy and mania, by compressing the carotid, and in some cases he instructed the patients how to perform this operation for themselves. ("Megrim and Sick-headache," pp. 276-7.)

On this theory also we can see at a glance how venous congestion produced by coughing, etc., may still further impede the outflow of blood from the intra-cranial veins and produce vertigo fits and other members of this group.

When my headache is bad it is simply impossible to assume the horizontal position, and when in bed I have to sit bolt upright against the back and slide gradually down if the pain gets less. I have also pointed out that by turning the face up so as to look at the ceiling the pain is somewhat relieved. It seems to me that this position may counteract to some extent the arterial inrush, especially from the vertebral arteries, and may also facilitate the outflow of venous blood by the jugulars, but I should like to hear the opinion of a more competent anatomist on these points.

There is probably no fact that is more widely recognised in this portion of the globe, than that mental irritability and depression are relieved by the smoking of tobacco, and if we look up tobacco in a system of therapeutics we find that it is classed with the depressomotors, *i.e.*, with the drugs that diminish muscular power and enfeeble the heart, and among drugs having a similar action it is interesting to note that ipecacuan and antimony have a similar power in relieving migraine. Now, how do they act? It seems to me that tobacco relieves mental irritability and depression, not so much by relaxing the arterioles, though its effect on the muscles may also do something in this direction, as by weakening muscle power generally, and especially that of the left ventricle of the heart, it thus reduces arterial tension and relieves the intra-cranial circulation, and we know that if tobacco is pushed beyond the stage of mental well-being it may cause nausea, and very serious depression or syncope.

My observations on the effects of tobacco smoking on the pulse seem to show that it lowers arterial tension, and quickens the rate of the heart's action, and it is easy to understand that in accordance with Grashey's observations it will in this way increase the volume of blood passing through the brain, and so produce well-being; and this well-being produced by tobacco is not accompanied by a desire for activity and a feeling of tone in the muscles, but rather by slight languor and disinclination for exertion.

I think also that it is of great interest to note that a physiological condition which has a similar effect on the heart has also a similar effect on the uric acid headache; I refer to old age, for it is well known that as sufferers begin to grow old this headache gets less frequent, or even entirely ceases.

The explanation is, I think, fairly simple, and I have been fortunate enough to see or hear of several cases of old people who have lost their headaches in old age, after being regular periodical sufferers all their lives. But a closer investigation of such a case brought out the following interesting facts: the headache indeed was absent, but the rest of the phenomena were not absent, just as for so many years before there came a time when the urine was scanty, followed shortly by a time when the urine was profuse, with the scanty urine went coldness of skin and extremities and some mental depression, some rise of arterial tension, but in place of a long slow pulse a fast and irregular one, and a palpitating heart.

This sufficed to explain everything; of uric acidæmia and an increased excretion of urates we had ample evidence—it was not that they were absent, but that their effect had altered.

In place of a long slow pulse we had a short and fast one and a palpitating heart, the heart was failing—no uncommon occurrence in old age—and owing to this failure the intra-arterial pressure, in spite of contracted and degenerate arterioles, was not great enough to compress the intra-cranial veins and produce headache; all the other factors were present as before, the heart and head signs alone had altered, and the alteration of the former explained the alteration of the latter.

In a similar way, and doubtless for similar reasons, mental depression and irritable temper often get decidedly less marked as old age advances.

As a rule in the young and middle aged, whose hearts are intact and well nourished, the severity of the symptoms is proportional to the amount of urate that is passing through the blood and being excreted in the urine.

Thus with an excretion of .6 gr. per hour, there will be mental irritability, mental and bodily inability and depression; with an excretion of .7 gr. per hour and above there will in the same person be headache or fits—whichever he happens to be liable to, and in practice we often see mental depression and irritability forming a sort of halo round the other more severe disturbances of function.

In my own case, though the amount of urate passing through my blood is now rarely sufficient to produce a headache, I much more often suffer from some amount of irritability and depression.

Another instance of the same thing may be seen in post febrile bradycardia and the depression which follows acute disease, due, I believe, to post febrile uric-acidæmia.

Writers on post febrile bradycardia (see "Uric Acid," pp. 117 and 135) have observed quite correctly that this condition is met with in its most severe forms in those who have been previously strong and healthy, and yet in apparent contradiction they attribute the bradycardia to weakness of the heart muscle.

Why should those who were previously strong and healthy have the weakest hearts after a fever? There is no difficulty in showing, however, that they have most uric acidæmia, and the post febrile phenomena are proportional to this.

In my book ("Uric Acid," p. 135), I have mentioned two cases of pneumonia that were under my care about the same time: one was a strong and powerful man, the other a weak and ill-nourished woman; in the former the post febrile conditions, including bradycardia, were very prominent, in the latter very slight, and the difference was simply due to the fact that the big man with his large metabolism of nitrogen stored much urate at the time of the fever, while the ill-nourished woman stored but little, and the post febrile symptoms were proportional in each case to the amount of urate passing through the blood (post febrile uric-acidæmia).

As regards the heart, the only necessary quality for the production of bradycardia is that it shall be sufficiently strong to withstand the strain which uric acidæmia, contracted arterioles, and high arterial tension put upon it, and this power is, to my mind, much more likely to be met with in those who were previously strong and robust.

If, on the other hand, the heart fails before the high tension, we get palpitation and a quick and irregular pulse marking the period of post febrile uric acidæmia, just as in the case of headache previously mentioned.

During our recent epidemic of influenza, I have several times been struck with the fact that the old and feeble, when they recover from uncomplicated influenza, have far less after depression than those in the prime of life, and I have no doubt that their small nitrogenous metabolism, as in the case of the woman just mentioned, accounts for this, and I have little or no doubt that the severe depression which follows influenza is simply a prolonged post febrile uric acidæmia, which, if not actively treated, may lead the way to more or less permanent feeble nutrition.

The old, again, are probably let off some of the post febrile mental depression because their hearts are unable to maintain the arterial tension necessary to interfere with their intra-cranial circulation, and their comparative immunity is thus due to two causes.

But the absolute dependence of all the phenomena on the amount of urate present can easily be demonstrated in physiological conditions.

Let anyone who does not mind a little auto-experimentation take, say, gr. xl. of an alkaline salt of sodium or potassium three times a day, and continue this for several days; during the first two or three days as the acidity falls the excretion of urates will rise, the urine will become scanty, and he will suffer from headache, mental depression, and coldness. Let him continue the same dose for another two or three days, he will find that the urine gradually becomes more profuse, and that the headache and mental depression disappear, and feelings of well-being take their place. But the alkali is still being taken. What is the alteration due to? He will find that the urate excretion has altered; it was at first very high, but after two or three days the immediately available stores were exhausted, and the excretion fell down nearly to formation level (*i.e.*, 1.33), and little or no urate passed through the blood. This is the cause of all the other alterations, the alkali itself having no direct effect whatever.

Or the experiment can be varied by taking salicylate of soda, gr. xv. quater for two or three days, and following it by the alkali as before.

The salicylate is a powerful solvent, and quickly clears out all the available stores of urate. The alkali given in sequence to it will cause but a comparatively small excretion of urates, and the symptoms it produced on the previous occasion will be more or less completely absent.

Having taken the view that, in accordance with the above facts, mental depression bears exactly the same relation to the excretion of urates that headache does, and that the causation of the one differs from the causation of the other merely in degree, I was much interested to be told by one of my patients (a man of conspicuous mental ability, who had all his life suffered from mental depression), that when he had his worst attacks, which were always in the early hours of the morning (note the parallel with the normal excretion of urates, which is always large in the early morning hours—early alkaline tide—and the urine scanty, though, as I have shown, there is plenty of water in the body, and no digestion is going on[1]), he was accustomed to obtain relief by sitting upright in bed in exactly the position that I have to assume in my headache ; and the fact is not only interesting as a parallel, but it proves, I think, that in this case the mental depression was not due either to feeble heart or to cerebral anæmia, and taken along with my argument about the effects of tobacco and cardiac depressants, explains the frequent association of high arterial tension with mental depression, because, as we now know, they are cause and effect.

I do not doubt that mental depression may be due to a weak heart, and consequent cerebral anæmia, but such depression would pay little or no attention to fluctuations in urate excretion, and would be benefited rather by lying down than by sitting up.

It is interesting also to remember that in mental depression associated with high arterial tension, digitalis does harm. I have several times given it in cases where there seemed to be a doubt as to the power of the heart, and have seen cases where others have done the same, and the only result was to make the depression worse.

One can easily understand why this should be so, as digitalis affects both factors in the causation of high tension: it strengthens the heart and contracts the arterioles. On the other hand, where mental depression is associated with real cardiac weakness and low tension, digitalis may do a great deal of good.

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[1] See also Proceedings of the Medical Society of the State of Pennsylvania, June, 1890, as to early morning hours in melancholia.

Talking of mental ability reminds me of a point of some interest in this connection.

Dr. Liveing speaks of the person whom he would expect to be affected with megrim as "a man of high intellectual attainments" (prev. ref., p. 35), and Dr. Wilks[1] considers that megrim is rather rare among the lower orders, and though I have seen a good number of cases among hospital out-patients, I do not recollect to have seen any among the lowest class, the ordinary day labourer.

There seems, then, to be some reason to think that megrim is more often met with among those who have naturally active brains, and if this activity has as its anatomical basis the possibility of a free supply of arterial blood, it is not difficult to see that those who have the largest arteries will be most likely to have their intra-cranial veins compressed when the intra-arterial tension rises too high.

I have mentioned above Grashey's supposition that one of the effects of increased intra-cranial pressure and stasis might be an increase of cerebro-spinal fluid which might further increase the pressure, or keep it up even after the arterial tension had fallen.

Now Dr. Broadbent,[2] whose opinion I have several times quoted before, considers that the phenomena of uræmia represent the effects of high tension on the circulation of the brain, and Grashey's researches make it easy for us to understand the way in which it acts.

But in accepting this explanation I have had considerable difficulty in understanding how it was that when, in some such cases, the temperature rose, this being almost certainly accompanied by a relaxation of arterioles and a fall in arterial tension, the cerebral symptoms, coma, &c., remained unaltered, *i.e.*, that the cause being removed, the effect still continued; but if one of the effects of the high tension is to bring about an increase of cerebro-spinal fluid, it is quite possible that so much pressure may be produced in this way, that the fall of arterial tension, due to the rise of temperature, is no longer sufficient to relieve the intra-cranial pressure and the coma it produces.

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[1] *Lancet*, vol. ii., 1888, p. 263.

[2] "The Pulse," p. 245.

The same fact may also explain another difficulty, for looking upon uræmia as organic uric-acidæmia, because its symptoms all resemble so closely those of functional uric acidæmia (see "Uric Acid," pp. 226-7), it was a surprise to me on examining some blood drawn by cupping glass from a patient who was dying of uræmia, to find little or no uric acid. The patient, however, had considerable fever at the time the blood was drawn, and fever, as we know ("Uric Acid," pp. 57 and 118), clears the urate out of the blood and relaxes the arterioles. The fact, therefore, that I found no urate did not prove that it had not previously been there, and we know that there is often a large amount in the blood in Bright's disease, it being, as I have suggested, unable to pass freely through an inflamed kidney, and Grashey's suggestion about the effect of high tension on the cerebro-spinal fluid may explain why the cerebral symptoms did not depart along with the uric acid, when the temperature rose.

Just as the uric acid headache corresponds with a fluctuation in the excretion of urates, so very often do the fits of epilepsy, and the mental condition in each case fluctuates with the urates, there being mental brightness and well-being with the minus excretion which precedes the attack, and more or less misery and depression with the plus excretion that accompanies the attack; and so constant is this that quite a number of cases have been recorded in which the sufferers were able to predict an attack of headache or fits, as the case might be, from the fact that they were feeling more than usually bright and well.

Then, again, in many cases, the fits appear, if we may take the word of the patient, to be very decidedly influenced for good by drugs such as acids, opium, mercury, salts of zinc, copper, silver, nitro-glycerine, &c., which diminish the excretion of urates and clear them out of the blood.

And as to diet, very numerous cases are recorded in which a vegetarian diet has effected a cure, and there is, I think, but little doubt of the way in which this diet acts in the parellel case of the uric acid headache.

I have now before me the notes of a case in which a gentleman, aged 50, who had suffered from fits all his life, was cured by a severely vegetarian diet, with a purgative dose of calomel and colocynth, followed by haust. Sennœ co. twice a week, and he lived twenty years more without any fits at all.

He lost weight very decidedly, and was considerably weakened by the treatment, and I think it probable that the urate formation was not only greatly reduced, but that the heart was also much weakened by the severe treatment, and hence was incapable of keeping up so much tension as was necessary to compress the intra-cranial veins.

Probably this treatment was unnecessarily severe; at the same time there are, no doubt, plenty of epileptics who would be glad to purchase freedom from their disease at even greater cost.

The treatment in the above case was recommended by Dr. Pelham Warren, long since dead, and the directions say, among other things, "the medicine is meant to remedy the mischief arising from an inactive state of the liver, indigestion, &c., but the constitution cannot be freed from the unhappy tendency to pressure on the brain and convulsions without the strictest persevering attention to diet."

"Pressure on the brain" is an extremely good and possibly accurate description of the real condition present, and it reminds us of what patients with headache or depression so often say—that their head is bursting, or their brains boiling over. We can now realise much more thoroughly the condition of affairs that underlies these sensations.

As to epilepsy, some interesting researches have been published by Drs. C. A. Herter and E. E. Smith (*New York Medical Journal*, August, 1892.)

In the publication of some previous researches on uric acid, these authors were inclined to take objection to my results on account of the process used; but since that, I and others have pointed out that Haycraft's process is an extremely accurate one (see *Journal of Physiology*, vol. xiii.), and that as regards most of my results, some very similar, if not identical, ones have been obtained by others with various processes.

As regards epilepsy, I have pointed out that the excessive excretion of uric acid which accompanies the attack is sometimes so obvious as to be visible to the naked eye as a deposit falling through the urine before it is cool (see "Uric cid," p. 87), and where the fit is well marked and the eparation of urine accurate, an excess of uric acid can be ound by almost any process.

As regards the remarks of Herter and Smith on epilepsy (prev. ref., p. 224), it is quite evident that in the case of grand mal they have not sufficiently separated the urine corresponding to the fit, and, as I have frequently pointed out before, it is quite useless to expect a positive result if this is not done.

The smallest amount of urine examined in their tables is 134 cc., and this was not at the time of an attack of grand mal. All the amounts estimated at the time of an attack of grand mal were larger than this.

Now the amount of urine excreted in an ordinary grand mal attack, lasting, including the stupor following the fit, 20 to 30 minutes, could not be much more than 30 to 40 cc.; therefore, even under the most favourable circumstances, the urine of an attack was mixed with two volumes of urine excreted before or after it, and no excess of uric acid would therefore be expected.

As I have pointed out, even in the case of the uric acid headache, the excretion of the whole day may show minus uric acid, while if the urine of the attack is carefully separated, it shows plus uric acid corresponding to the hours of headache (see *Journal of Physiology*, prev. ref.). In my case of epilepsy published in the *Neurologisches Centrallblatt*, to which the authors refer, the urine of each fit was separated as carefully as possible; and I also pointed out that when the urine of the fits was mixed with that before and after them, the uric acid came out normal, while the urine secreted during the fits showed a great excess.

In other cases I have got patients to pass water every hour of the waking day; then, when a fit occurred, we had the urine of the hour preceding the fit ready for examination, and the bladder never contained more than one hour's urine in addition to that of the fit, and this, in my opinion, is the way in which a uric acid reaction should be looked for in grand mal. Certainly it is quite useless to examine 150 to 500 cc. of urine when that excrete during the fit could not possibly exceed 20 or 30 cc.

The authors say again on p. 234: "The latter [that i urines passed immediately after seizures] are apt to sho a higher uric acid ratio than the urines passed just befor seizures; very often, however, the ratio is one that belongs within the limits of health."

This is just my result diluted, and as the quantities are not given there is nothing to show that the urine just before was not the urine of two to three hours before, *i.e.*, a mixture; or that the urine passed after the fit really corresponded only to the excretion of the fit.

Further down on the same page they say: "The excess of uric acid that is observed in epilepsy cannot reasonably be construed as the cause of the seizures." They acknowledge, then, that there is an excess, but differ from me as to the relation of cause and effect.

In petit mal, however, they found a distinct relation between the excretion of uric acid and the cause of the seizures, and treatment by milk diet greatly reduced the frequency of the attacks (prev. ref., p. 239.)

This is just what I should expect, as in my writings on the subject I have not limited myself to grand mal, but have suggested that petit mal, hystero-epilepsy, and hysteria are all similarly related to uric acid (see "Uric Acid," p. 87 and 123).

Where attacks of petit mal are frequent I should expect a continuous plus excretion of urates; but, as I have often proved, it is useless to look for urate reaction in a single short attack, as the separation of urine can never be sufficiently good (prev. ref., p. 87).

If Herter and Smith will separate the urine of grand mal cases with greater precision, I have no doubt that they will get urate reactions similar to mine, and if they will also treat grand mal to the same diet as petit mal they will, no doubt, get some satisfactory results, just as others have already done.

The observations of these authors on the relationship of the fits to intestinal putrefaction are of extreme interest because they supply a further illustration of a point which I had previously noticed. Thus, in St. Bartholomew's Hospital Reports, vol. xxiv., p. 226, I have made the following remark: "Salicylates again may fail to be absorbed for a time from gastric irritation or other causes, and I had a remarkable instance of this in a case of epilepsy which I was treating with this drug. On several occasions the urine gave no reaction with perchloride of iron during the fits, though on some occasions there was probably as much as 20grs. of the salicylate lying in the stomach, and there was a strong reaction in the urine after the fits had passed off, without any more of the drug being given."

I can now explain all the facts much more completely than I could at the time the above was written, for I now know that when uric acid in the blood raises arterial tension it contracts the arterioles and capillaries throughout the whole body; so that not only are the urine and saliva scanty, not only is the skin cold and dry, but all the gastro-intestinal secretions (gastric juice, bile, pancreatic and intestinal juices), are similarly diminished, and the circulation through their mucous membranes is so imperfect that digestion and absorption come to a standstill. This and the generally diminished metabolism accounts for the marked fall in urea excretion that generally accompanies a plus excretion of uric acid, and the resumption of these physiological processes accounts for the rise of urea when the urates are cleared out of the blood, and their excretion in the urine diminished.

But such a marked arrest of gastro-intestinal digestion will almost certainly be accompanied by the onset of putrefactive processes—the processes of which Herter and Smith have observed some of the results.

All is now clear; the reason the salicylate failed to appear in the urine in the above case was because the arterioles were contracted, and gastro-intestinal digestion and absorption were at a standstill.

We can now see clearly the origin of the horrible fœtid dejecta often met with after an attack of dyspepsia, bilious attack, or uric acid storm; also why calomel so promptly puts matters of this kind right. I used to think that it acted directly as an antiseptic, but it is now evident that its most important action is the result of its chemical relations to uric acid, by which it clears urates out of the blood, relaxes the arterioles and capillaries, and allows gastro-intestinal digestion to proceed once more.

The intestinal putrefaction noticed by Herter and Smith, far from being the cause of the fits, is but a result of uric-acidæmia. The contraction of arterioles which produces the high tension, which in turn interferes, as I have pointed out above, with the intra-cranial circulation and produces the fits, interferes also with gastro-intestinal digestion and allows putrefaction to take its place.

Mental depression, again, is influenced by diet and drugs in just the same way as the above troubles; there are plenty of recorded cases in which a vegetarian diet has done good or effected a cure.

Hysteria is another instance of the same thing; its attacks bear the same relation to the excretion of urates as do those of the above-mentioned troubles, and, like these, it can be relieved by acids and other drugs which clear urates out of the blood; and I have suggested that the fluctuations in the excretion of urea that accompany and follow such attacks[1] represent the effects of uric acidæmia on the metabolism, for uric acidæmia, however produced, is accompanied by a diminution in the excretion of urea, which I have suggested is due to a diminished metabolism, the result of the general vascular contraction which the uric-acidæmia produces; and when the uric-acidæmia is put an end to, whether by drugs or by natural causes, and whether it has produced headache, mental depression, hysteria, or fits, the relaxation of arterioles is quickly followed by a rise in the output of urea (see "Uric Acid," p. 122-4).

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[1] See Researches of MM. Gilles de la Tourette and Cathlineau in the *Progrès Medical*, vol. i., 1889.

We can now see that the relation of hysteria to the uterine functions, and especially to menstruation, is due to the fact that this, even in physiological conditions, is almost certain to bring about a fluctuation in the excretion of urates, and a temporary uric-acidæmia, which, in turn, may produce hysteria, or any other members of the group, by its effects on the intra-cranial circulation. (See BRAIN, 1891.)

These considerations seem to me to warrant the conclusion that in none of these so-called neuroses does the disturbance of function ever originate in the nervous system, which would work on with the utmost regularity if its circulation was not disturbed.

But we can also see quite clearly that intra-cranial lesions, whether traumatic, syphilitic, or of other origin, may have a powerful influence in hindering or increasing the effects of uric acidæmia on the intra-cranial circulation.

As I have said before with regard to headache, it is quite unnecessary to treat the nervous system at all if the uric acidæmia is prevented, and it appears to me to be quite evident that many of those who have believed that they were treating the nervous system direct, have really been treating the urid acidæmia unknown to themselves; for such drugs as morphine, the nitrites, the iodides, the salts of zinc, copper, silver, and mercury, have a powerful effect on the excretion of urates, and tend to remove uric-acidæmia and its effects on the intra-cranial circulation; and even the bromides with their depressant action on the muscles and the heart, may, like tobacco (mentioned before), affect the intra-cranial circulation rather than the tissues of the brain direct.

It is evident, then, that we can not only completely account for the relation which these functional disturbances have been found to bear to a large excretion of urates, and can see that this depends on their both being the results of one common cause—an excess of urate in the blood; but the explanation places us in a more favourable position than we have ever previously attained for making an attack on the real *fons et origo mali*.

In conclusion, I will shortly mention one or two points in treatment.

It is impossible to prevent fluctuations in the excretion of uric acid; they occur in every one from hour to hour; therefore the only way to prevent severe uric acidæmia is to diminish the formation of uric acid.

A wave 10 feet high at low tide may do little or no damage, while a wave of similar height at high tide may sweep everything before it. We must keep the uric acid tide always low, and then its waves will do no damage.

It appears that uric acid is generally formed at or about the proportion of one grain for every 33 grains of urea; therefore we can take urea as our guide, and, reducing its formation, can calculate that of uric acid from it.

If a patient suffers from headache or mental depression, and passes on ordinary diet 600 grains of urea per day (*i.e.*, forms 18 grains of uric acid), let him try the effect of such diet as will reduce his urea and keep it about 400 grains per day (*i.e.*, forming 12 grains of uric acid), and such a diminished output of urea may be effected without either starvation or loss of weight.

It seems also to be advisable to avoid meat which contains much urate already formed, also soup, beef-tea, and meat extracts, which are, as I have shown, very rich in urates (see "Uric Acid," p. 51).

In epilepsy it may be necessary to go further, to reduce urea still more, and even to produce considerable loss of body weight, as in the case I have mentioned.

I have great hopes that by careful advance in the direction which these facts indicate, we may eventually obtain a really valuable power of control over these functional disturbances.

Critical Digest.

ON RECENT APPLICATIONS OF GOLGI'S METHOD TO THE STUDY OF THE NERVOUS SYSTEM.[1]

BY WILLIAM ALDREN TURNER, M.B., M.R.C.P.

THE examination of the minute anatomy of the central nervous system by Golgi's staining method, which has brought forth much valuable information at the hands of Golgi, Kölliker, Ramón y Cajal and others on the Continent, has as yet received but scant attention in this country. It is therefore proposed to give here a *resumé* of the more important papers which have appeared on this subject, the literature on which is now voluminous.

The *method* of staining, although modified in details by various workers, is briefly as follows :—Small portions of the central nervous system of embryos are placed in a mixture composed of bichromate of potash (4 parts) and perosmic acid (1 part). After remaining in this solution for several days, the blocks of tissue are washed in a quarter per cent. solution of nitrate of silver and finally placed for two days in a three-quarter per cent. solution of this salt. They are then ready for imbedding either in paraffin or celloidin. However carefully done, the method has its disadvantages, which are, that all the nerve structures are not equally and at the same time stained, so that the relation of the different elements, *e.g.*, nerve fibres and nerve cells, is not always evident.

Kölliker finds that the nerve fibres and their branches stain most readily, so that one sometimes sees sections in which, to all appearance, there are no nerve or glia cells.

The *theory* of the action of this staining method has provoked discussion. It is held by some that the black staining which is characteristic of it is due to the precipitation of bichromate of silver in the pericellular lymphatic vessels; the reasons for this statement being that the cells are larger than where observed by other methods, and that their shape is different; that ammonia dissolves the deposit revealing a smaller cell, and that the lymph channels in other organs are also stained by Golgi's method. This view is not accepted by all in its entirety, for Kölliker believes that both a staining and a precipitation takes place. If the elements present a sharply-defined smooth contour, it is indicative of staining, if they are irregular and varicose, precipitation has occurred; the former is chiefly observed in non-medullated fibres, the latter in neuroglia cells. Ramón y Cajal inclines to the view that the reaction is due to staining and not to precipitation: he has never obtained deposits in the lymphatic spaces of the cornea, lymphatic vessels or conjunctival lacunæ, while he has constantly seen the cells in epithelium, cartilage, and muscle stained by this method.

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[1] This has already appeared in a slightly modified form as a series of papers in the *Journal of Anatomy and Physiology*, vols. xxv. and xxvi.

This preliminary introduction upon the method is now followed by a consideration of the anatomy of the central nervous system, which has been obtained mainly from the works of Golgi, Ramón y Cajal, Kölliker, and Van Gehuchten. A bibliography at the end summarises briefly the important works on the subject.

A. SPINAL CORD (KÖLLIKER).

1. *The posterior roots and posterior columns.*—The posterior nerve roots enter the cord by numerous small bundles which gently ascend in the postero-lateral groove; and turning mesially towards the posterior columns, each fibre divides into two portions, an ascending and a descending. In men and pigs this division takes place at angles of 150° or 160°, and as the fibres of a root bundle split at the same level, and the divisions retain their oblique course for a long time, the superficial portions of the posterior column present a peculiar and characteristic arrangement of fibres. In their further course these fibres extend into the deeper parts of the column and become longitudinal fibres. When examined more closely, the posterior roots are found to sink into the region of the lateral segment of the substantia gelatinosa in the part of the posterior column recently called the border tract (Randzone) of Lissauer. Here the root bundles divide into two systems, a smaller lateral one, which passes towards the postero-lateral corner of the substantia gelatinosa, and a stronger, stouter median one, which courses in the border tract towards the posterior column. In the lateral system all the root fibres divide at the same place; in the median they diffuse over the whole space between the entrance of the roots and the posterior column, occupying in antero-posterior diameter the ventral

part of the border tract, while the dorsal portion is formed by a border of longitudinal fibres. The divisions are here scattered over a considerable area; and the ascending and descending fibres which are given off from these so intertwine amongst each other, that in cross sections a very delicate alternation of small bundles of fibres exists. As a result of his investigations on the spinal cords of human embryos, as well as on those of sheep and new-born kittens, Kölliker is inclined to believe that all the sensory root fibres divide.

It is important to consider now the further course of the branches of the "sensory" roots, and to find out definitely how they end. The posterior columns, by which are meant not only the usual so-called columns, but also the connected layers of white matter which are situated outside the substantia gelatinosa and connect the posterior and lateral columns, are essentially composed of the fibres of the posterior roots.[1] The longitudinal fibres of the posterior columns bend at a right angle and pass into the substantia gelatinosa, where they break up into fine branches (in the spongy substance), and behave in a manner similar to what is later on described as occurring in the "collaterals" of the column fibres. These bendings are not abundant all over, and it is impossible to say whether they are only in the descending or also in the ascending fibres. Some are true terminations, and divide in the posterior horn into delicate branches, others are longitudinal fibres which turn back in the substantia gelatinosa by another bending to their former course. It is difficult to say how many of the branches of the posterior root fibres pass into the grey substance to end there, and how many pass up to the medulla oblongata. It is most probable that all the descending branches pass into the grey substance, and that the ascending mostly pass up to the brain. Kölliker believes also that these latter may partly enter the grey substance, as he has observed in longitudinal sections that some of the fibres entered the grey matter at right angles and ended there. Observation of many sections further shows that a great number of the fibres of the posterior columns run a very considerable distance without interruption.

It is of interest to notice if this method throws any light upon the vexed question of the origin of the "sensory" roots in cells of the grey matter of the cord. The observations of Golgi, Ramón y

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[1] It is essential to remember that the posterior horn and substantia gelatinosa is of extraordinary breadth and thickness in the embryo. As growth proceeds, the grey matter, which at first prevailed, gradually gives place to the white.

Cajal, and Kölliker cannot confirm this statement; none of them has ever seen a nerve cell passing into a "sensory root" fibre. Until further proof is forthcoming for the higher animals, it is well to teach that the origin of the "sensory" root fibres in the cord cannot be held as proved.

2. *The Collaterals* (*Collateralen*) *of the Posterior Roots and Posterior Columns.*—The existence of lateral branches (collaterals) of the longitudinal fibres of all the columns of the spinal cord is a most important discovery of Golgi and Ramón y Cajal. They are very readily seen in longitudinal sections, and are a noteworthy feature of the minute structure of the cord, and form one of its most important physiological relations. As a rule they are not so thick as the longitudinal fibres, and like them they present varicosities, especially on their fine ramifications and at their terminations, which, according to Kölliker, are artificial. The collaterals are usually given off at acute angles, but in some cases they arise from the stem fibre at a right angle.

All collaterals end in small *terminal bushes or tufts,* which remind one of simple end plates. These small tufts consist of numerous short twigs of the finest varicose fibrils, which encircle the bodies of nerve cells, and mainly end in fine knobs. When the cells are large, as in the anterior and lateral horns, these twigs form very beautiful pictures, and one notices that there are no anastomoses of neighbouring twigs, and that they are not in direct association with the cells. Similar endings are found in the substantia gelatinosa, substantia spongiosa, and in Clarke's column. As at their terminations the collaterals show no sign of anastomosis in their course.

We will now consider in detail the collaterals of the "sensory" roots and their branches and of the posterior columns. After these roots have entered the border tract, they give off in certain cases before their division single lateral branches. Similarly the fibres of the border tract and posterior column give off collaterals. The number of collaterals given off is very great, varying from two or three up to nine in very long fibres, and they follow each other at greater or less distance. They occur in all parts of the cord, both in the limb enlargements, in the intervening portions, and in the upper cervical region, and so many are there that it is difficult to see a fibre which does not give them off. In all portions of the posterior columns, superficial, middle, and deep collaterals are present; it is therefore reasonable to conclude that the majority of the fibres of the posterior column give off collateral branches. It is, however, worthy of note that in many preparations the collaterals are less well or not at all stained. In the dorsal corner of the posterior column of the cervical cord of a calf embryo, Kölliker noticed a small compact bundle which possessed no collateral fibres, while they were well formed in all the other portions of the column.

The sensory collaterals, as the lateral branches of the posterior column fibres may be called, pass from the different portions of the column into the grey matter of the posterior horn, and course more or less towards its ventral or motor parts, in order to terminate in its different regions, and even in the anterior horn of the same side. Their passage into the grey matter is in bundles of five, ten, fifteen, or more fibres, which enter partly in a straight course, and partly arched and slanting through the substantia gelatinosa. The place and nature of their termination is important—

(1) In the substantia gelatinosa in the manner described above as characteristic of collaterals.

(2) In the boundary layer between the substantia gelatinosa and spongiosa; here the fibres are numerous, and form a delicate felted arrangement.

(3) In Clarke's column, where they form a network similar to that in 2.

(4) In the substantia gelatinosa of the opposite side. In some young animals a posterior commissure is found to exist and to be composed of collaterals. Ramón y Cajal describes the posterior commissure in new-born dogs as consisting of three parts: anterior and posterior arching bundles, and a middle transverse portion. The anterior bundle has its concavity towards the central canal, and consists of delicate and coarse fibres, which spread out laterally in all directions. The origin of these fibres is doubtful. The posterior bundle has its convexity to the ventral side, and consists of the collaterals of the posterior columns, which end in the opposite side in the adjoining portions of the substantia gelatinosa. The middle (a cross-running bundle) goes midway through Clarke's column, and ends in the lateral part of the substantia gelatinosa. The elements of this part of the commissure arise partly in the collaterals of the lateral columns, and partly their origin is doubtful.

(5) In the substantia spongiosa of the posterior horn of the same side.

(6) In the anterior horn. There is a very important group of the sensory collaterals which penetrate the anterior horn of the same side, called by Ramón y Cajal the "antero-posterior" or

"sensori-motor" group. Springing partly in the posterior column and partly in the border tract, they collect in the substantia spongiosa into stout bundles, and penetrate as compact strong fibres straight into the anterior horn, where they spread to all parts, and end in the region of the nerve-cell groups in the finest branches. These are defined by Kölliker as the reflex collaterals of the sensory roots.

3. *Collaterals of the Anterior and Lateral Columns.*—The collaterals of the lateral column fibres pass partly into the ventral portion of the posterior column and of Clarke's column, and partly into the anterior horn to the various motor cell groups. Some single fibres pass into the boundary region between the substantia gelatinosa and spongiosa and towards the white commissure. The collaterals of the anterior columns all pass dorsalwards, some cross into the commissure, others towards the anterior and lateral horns of the same side, while others still go into the posterior columns. These latter form end bushes, which remind one of the long sensory collaterals, and are most perfectly recognised in sagittal sections. Concerning the end ramifications of these collaterals, Kölliker found, what he had not previously noticed in the sensory ones, long dependent branchlets, which give off on both sides, at nearly a right angle, a great number of short branches, which end as above described. Many of these collaterals only show such divisions, but others are in all respects similar to the sensory ones.

The anterior commissure consists partly of axis cylinders, which decussate, and partly of collaterals of the anterior and lateral columns, which often show branchings in the commissure and end in the grey matter of the opposite side, where they partly spread out into the anterior horn and partly turn toward the posterior horn. It is not impossible that among the last-mentioned fibres posterior column collaterals exist, which pass through the commissure to the opposite side.

4. *Nerve Cells.*—The cells of the grey matter of the spinal cord are all multipolar, but differ essentially, in that (1) some possess one unbranched or only sparsely-branched nerve process, (2) while in others this gives off numerous branches; this latter set are again divided into two sub-classes: (*a*) those in which the axis-cylinder process, in spite of its giving off branches, does not lose its individuality; and (*b*) those whose axis-cylinder process divides into delicate and very fine ramifications. According to Golgi's classification, the cells of the first category and of (2) (*a*) are motor, while those of (2) (*b*) are sensory.

Another classification of nerve cells is based on physiological grounds, and is as follows :[1]—(*a*) *Motor Cells,* which pass into the motor root fibres or give origin to the same. These cells lie in the anterior horns and form nerve-cell groups; they are as a rule large, but may be small, especially in the median part of the ventral segment of the anterior horn, and in the region between the anterior and posterior horns. The nerve process of these cells goes usually straight towards the exit places of the roots, but others are arched. It is most probable that these fibres are not branched, although Ramón y Cajal mentions that he once saw in a rat a lateral branch of such a fibre coursing backwards. Golgi also describes such. It is yet doubtful whether these are constant or only in single cases. This point is of great importance physiologically, and the simplest explanation of such a branch is that a motor cell may in certain cases branch out into centrifugal fibres.

(*b*) Cells whose nerve process is transformed into a longitudinal fibre of the white matter [Strang-zellen—column fibre cells]. They can be sub-divided into (1) cells whose process remains on the same side of the cord, (2) cells whose process passes by the white commissure on to the opposite side [Kommissurenzellen of Ramón y Cajal]. The great majority of these cells send their nerve processes into the lateral columns, many also into the anterior; the former lie mainly between the central canal and the projecting median corner of the lateral column; the latter are especially related to the median cell group of the anterior horns. The nerve processes of these cells partly possess no branches; in many cases, however, they give off a great number of branches without losing their individuality. The manner of ending of the stem fibre of the nerve process is noteworthy. In the majority of cases, as Golgi first saw, the end of a stem fibre bends nearly at a right angle into a column fibre. Kölliker considers all as nerve fibres which bend out of the longitudinal columns into the grey matter at a right angle, especially if they show no diminution in their diameter, and if they cannot be followed up to a nerve cell. Secondly, the stem-fibre ends by dividing, forkwise, into ascending and descending column fibres, just as the sensory roots do at their entrance. And, lastly, there remain a few processes, whose relation in the white substance is not yet settled, whether they pass away in one or in divers directions.

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[1] The examination of the axis-cylinder processes of the cells of the cord is one of very great difficulty, owing to the fact that by the Golgi method, as already pointed out, all the cells are rarely stained.

According to Golgi and Ramón y Cajal, all the nerve processes of this category pass into column fibres. In a preliminary communication Kölliker believed that those which crossed in the anterior commissure passed into the anterior roots of the opposite side. As he has been unable to find a relation between the commissure fibres and the nerve roots, he has no reason to hold his opinion any longer.

(*c*) Cells whose nerve processes give off branches right up to their terminations, but do not pass beyond the grey matter. These were first described by Golgi, and appear to exist only in the posterior horns, but not exclusively. They are most marked in the neighbourhood of the point of the posterior horn, but have also been found at the dorsal border of the substantia gelatinosa and in other parts.

Especially important for the interpretation of these cells was the nature of the termination of their branches. Golgi and Ramón y Cajal noted that they ended free and with varicose branches; none of them end in tufts or bushes, as is so characteristic of collaterals.

For accounts and figures of the manifold forms of nerve cells and of their protoplasmic processes, the reader is referred to the works of Ramón y Cajal. It is necessary, however, to say that it is very difficult to define the protoplasmic processes of these cells. When one remembers that these branches are perfectly smooth, the pointed and granular appearance which they present, and which makes them resemble neuroglia cells, is probably artificial. Regarding these processes the following may be noted:—in silver preparations they form numerous coarse ramifications, so that in the organs of the new-born many cross through the whole breadth of the grey matter, and occupy half of its antero-posterior diameter. The median group of cells of the anterior horn are peculiar in that they send a portion of their branches through the anterior commissure to the opposite side—a relation which Ramón y Cajal first pointed out, and which Kölliker finds also in the cells in the neighbourhood of the posterior commissure. Some protoplasmic processes enter the white columns, and often penetrate right through to the surface of the cord.

5. *Neuroglia Cells.*—These consist of the elements of the original medullary plate, and are divided into primary and secondary groups. The first appear as the epithelium of the central canal, and originally pass through the whole cord, but later on are only present in the deeper parts; the latter arise later in all parts of the grey and white substance, likewise from the elements of the medullary plate.

Summary.—1. The sensory root fibres divide at their entrance into the cord into an ascending and descending branch.

2. Some of these sensory fibres course for a considerable distance (4-6 cm.) longitudinally; others arch into the grey matter, and end free in delicate ramifications.

3. An association between the sensory root fibres and the cells of the grey matter of the cord has not up to the present been observed.

4. All sensory longitudinal column fibres give off delicate lateral branches—the so-called *Collaterals* of Ramón y Cajal, which, entering the grey substance, ramify in all its parts and end free.

5. The motor root fibres arise from greater and smaller nerve cells of the anterior horn in a single nerve process, which in certain cases (always, according to Golgi), give off lateral branches.

6. The anterior and lateral columns consist partly of fibres, which are given off by nerve cells in all parts of the cord. These column fibre cells (Strang-zellen) give off from their axis-cylinder process a greater or less number of lateral branches into the grey substance, which end free.

7. The relations of the nerve processes of the anterior and lateral column cells to the fibres in the white substance varies. In some cases they simply bend upwards and pass into a column fibre; at other times they divide into two or three branches; or, again, such a nerve process splits into an ascending and a descending fibre, or apparently attaches itself laterally to a column fibre (the T-shaped fibres).

8. The great majority of the fibres of these columns give off collaterals, which enter the grey matter of the anterior horn and of the anterior part of the posterior horn, and there end free, just as the sensory collaterals do.

9. The anterior and lateral column fibres bend in many cases at a right angle into the grey substance, and there end free.

10. All collaterals of column fibres, and all lateral branches of axis-cylinder processes end in the same way. They give off at acute or right angles a greater or less number of branches, and finally form delicate end bushes, which encircle nerve cells without associating themselves with them on forming any anastomoses.

11. The anterior commissure consists of:—

a. Axis-cylinder processes of nerve cells of the grey matter, which, after decussating, are continued as longitudinal fibres of the anterior and antero-lateral columns.

b. The decussating collaterals of the anterior and lateral columns.
c. The decussating protoplasmic processes of a part of the median cells of the anterior horn.

12. The posterior commissure consists of:—
a. The decussating collaterals of the sensory root fibres.
b. Possibly of the decussation of the collaterals of the posterior part of the lateral columns.
c. Decussations of the axis-cylinder processes of cells placed laterally to the central canal and substantia gelatinosa (Ramón y Cajal). But this is very doubtful.
d. Decussations of the protoplasmic processes of cells of the posterior horns. This also is not quite ascertained.

13. The nerve cells are divided into:—
a. Motor cells (*vide* under 5).
b. Column cells (*vide* under 6).
c. Cells, whose axis-cylinder process does not go beyond the grey matter, and forms delicate ramifications in this. These only exist in the posterior horn.

14. The value of the size, shape, and character of the nerve cells is not fully known. It is at any rate certain that large nerve cells are not limited to the motor sphere only.

15. The protoplasmic processes of all nerve cells ramify over a great extent in all directions, often penetrate the white matter, give origin to no nerve fibres, and do not anastomose.

B. THE CEREBELLUM.

1. *Purkinje's Cells.*—Golgi showed the excessively rich ramifications of the so-called protoplasmic processes of these cells, and proved that they formed no anastomoses with each other. He believed that the most superficial of these branches were attached to the connective tissue at the margin of the molecular layer and to the vessel walls. He further made the very important discovery concerning the axis-cylinder processes of these cells, viz., that they gave off a certain number of lateral branches, which partly coursed back into the molecular layer and partly spread out into the granular layer.

2. *The Small Cells of the Grey Molecular Layer.*—Contrary to the statements of many authors, this method definitely showed that these cells existed throughout the whole thickness of this layer in not inconsiderable numbers, and possessed, besides

anastomosing branches, an axis-cylinder process which formed very delicate ramifications and coursed in very diverse directions, amongst which the horizontal was especially conspicuous. The end of this process was not seen.

3. *The Small Cells of the Granular Layer.*—Golgi first definitely pointed out that these were nerve cells. Their axis-cylinder processes are very slender, still lateral branches are occasionally found, and anastomoses of these processes with nerve fibres. The protoplasmic processes only divide very sparely, are short, and appear to end in small granule heaps (Körnerhaufen).

4. *The Large Cells of the Granular Layer.*—In human beings these exist as spindle-shaped bodies, and are characterised by the extraordinarily numerous ramifications of their axis-cylinder processes.

5. With regard to the *medullated nerve fibres*, Golgi showed that many ramified in the white matter, but especially in the granular and molecular layers, so that their very terminations showed a thick plaiting, the details of which it is impossible to follow out. These anastomose with—(1) The axis-cylinder processes of the small cells of the granular layer; (2) the lateral branches of those of Purkinje's cells; (3) the nerve processes of the small cells of the molecular layer. As the most characteristic part of this plaiting Golgi portrays those parts which lie at the boundary of the granular and molecular layers, and which present many coarse and fine horizontal elements.

Next to those of Golgi come the important observations of Ramón y Cajal, who used the rapid hardening method with bichromate of potash, osmic acid, and silver. The most important of these observations are briefly as follows:—

1. *The Small Nerve Cells of the Stratum Granulosum.*—The branched processes of these cells end in small tufts of closely-set short branches; the axis-cylinder processes ascend, without exception, into the molecular layer and form in this delicate longitudinal elements, running parallel to the convolutions. In this layer they divide at different levels at right angles into two longitudinal fibrils, .2 to .5 micromillimetre long, which after a course of .2 to 1 millimetre end free without branching.

2. *Small Nerve Cells of the Molecular Layer.*—The most note-worthy facts discovered by Ramón y Cajal upon these elements are that the axis-cylinders of all the cells lying near the granular layer form peculiar branches or baskets which encircle the bodies of Purkinje's cells and form holes for them.

3. In the cerebellar cortex he found also specially branching fibres, which passed from the granular into the molecular layer and which he divided into three kinds:—

(*a*) Fibres which passed from the granular into the molecular layer and ended there in a branched expansion. He was able to follow these fibres in some cases into the white matter.

(*b*) Fibres which, arising in the white substance, ramified in the granular layer, and showed in their course mossy appendages. These are the "Fibras musgosas" of Ramón y Cajal. The very terminations of these fibres, each of which possesses from 20 to 30 branches, are on the level of Purkinje's cells, and appear to pass over into longitudinal fibrils. He is inclined to construe these fibres as the sensory terminations of the axis-cylinders of peripheral nerves, and bases this hypothesis on these two facts:—(1) The optic nerve in birds ends in the lobus opticus in free branches; and (2) the sensory root fibres in the cord course in a similar way.

(*c*) Fibres which, ascending from the granular layer, end in nests which encircle Purkinje's cells. These are the "Nidos cerebellosos."

4. Concerning the cells of the cerebellum described by Ramón y Cajal, in addition to those facts mentioned above, are the following:—

a. The axis-cylinder processes of Purkinje's cells have processes which pass back into the molecular layer.

b. The axis-cylinder process of the large cells of the granular layer is extraordinarily richly branched.

5. Ramón y Cajal mentions no branches in connection with the medullated fibres of the cerebellum. Concerning the question which of the fibres (which are shown by Golgi's method) are medullated, he surmises that possibly the very terminations of the "fibras musgosas" may be, and also the branches of the axis-cylinder process of Purjinke's cells which bend into the molecular layer, for both of these sets may become the medullated longitudinal fibrils of the molecular layer. As non-medullated, Ramón y Cajal considers the axis-cylinders of the granular cells, those of the small cells of the molecular layer, and of the larger granular cells.

Since the recent observations of Ramón y Cajal upon the cerebellum have not yet been confirmed, and even those of Golgi have only been re-examined by a very few, it will not appear superfluous if I communicate the results of Kölliker's own observations upon the minute structure of the cerebellum.

1. *The Small Nerve Cells of the Granular or Rust-coloured Layer, or the Small Granular Cells,* (Körnerzellen).—The examination of these cells has only been made in cats, and in preparations which have been treated both by Golgi's long method and by the short bichromate of potash, osmium, and silver method. In both forms of preparations, but especially in the first, these nerve cells were found, as Ramón y Cajal described, to possess short protoplasmic and long axis-cylinder processes. The former averaged from 10-40 micromillimetres in length, were simple, and appeared to end in short branches; the latter sprang usually from one of the other processes, only seldom from the body of the cell, and penetrated with a serpentine course outwards into the molecular layer. In such preparations only single small granular cells are stained, so that they are easily followed in their entirety; yet one finds places at which two, three, or more cell-bodies are grouped together, and from which a great number of typical nerve and ramifying branches go off. It is not difficult to follow the fragile nerve-processes right into the molecular layer, and to prove that they are here transformed into longitudinal elements after they have divided into two branches, each of which forms a right angle with the stem. These longitudinal fibrils here and there apparently end free; but it ought to be emphasised that the free endings are possibly not such, for it is impossible to say whether the silver has stained them throughout their whole length. The number of these fibrils is very considerable, and longitudinal, oblique, and tangential sections of the convolutions show them in the form of a very thick and delicate striation of fibrils, some of which are varicose and tortuous, and pass through the whole thickness of the molecular layer to its most superficial parts. On cross sections of the convolutions these elements appear as fine dots or streaks, which fill in the intervals between the ramifications of Purkinje's cells.

In exceptional instances this longitudinal fibre-system has special relations. Kölliker agrees with Ramón y Cajal, firstly, upon the presence of branches to the oblique stem fibres within the molecular layer, which become longitudinal fibrils; and, secondly, upon the appearance of ostensible stem-fibres, which pass over at a right angle into only one longitudinal fibre. Ramón y Cajal thinks that such a fibre could represent the termination of a longitudinal fibre, and, since he never saw it in association with a small granular cell, but followed it well up to the white matter, could pass into a medullated fibre.

The following facts may be added regarding these longitudinal fibres:—They stain most readily in the innermost parts of the molecular layer, and seldom throughout its whole thickness. Further, the staining is only partial, so that, between large and small bundles, streaks exist in which none of the fibrils are stained.

2. *The Large Multipolar Cells of the Rust-coloured Layer.*—These cells which have been discovered by Golgi have been confirmed by Ramón y Cajal. Their nerve processes form very rich ramifications. Kölliker saw them in cats and in the embryos of pigs and puppies, and, in contrast to those just described, he calls them the large granular cells. They are far smaller than Purkinje's cells, and lie either at the outer margin of the granular layer, partly almost on a level with Purkinje's cells, and partly in the middle of the layer, or even at the inner boundary up against the white matter. From an angular cell body there pass towards different parts three up to six or more branching processes, which course partly between the granules and partly pass into the molecular layer, and can often be followed far outwards in this layer; but besides they can penetrate into the white matter, as was once observed in the deeper parts of the cerebellum.

3. *The Nerve Cells of the Molecular Layer.*—The nerve cells of this layer are divided into two sets, the large and the small, of which the former are situated in the deeper and the latter in its superficial parts. While this last possess the usual form of multipolar nerve cells, the former present a very strange disposition of nerve processes, which, as Golgi and Ramón y Cajal pointed out, course transversely for a certain distance in the region between the molecular and granular layers. This transverse fibre gives off, at right angles, processes which encircle as in a basket the bodies of Purkinje's cells. The bodies of the cells measure from 20 to 25 micromillimetres, and are usually drawn out longwise, and present four, five, or more angles. Besides the nerve process, they give off a large number of protoplasmic processes, which, mainly passing outwards, ramify in the usual way, and extend as far as the outermost parts of the molecular layer. The so-called nerve process of Ramón y Cajal is an altogether peculiar structure. Beginning very delicately at the body of the cell, it becomes in its horizontal and transverse course twice or four times thicker, and stretches over the bodies of Purkinje's cells in order to give off branches from place to place. These branches pass off obliquely, sometimes with a coarse, sometimes with a delicate root, and end in such a way that each branch or branchlet is transformed into quite a tuft or pencil of terminal fibres, which

encircle basketwise the bodies of Purkinje's cells.[1] To unravel them further is a work of considerable difficulty, but it is probable that the branches have no association with each other, but end free. Likewise their relation to Purkinje's cells is not easy to ascertain. From transverse and longitudinal sections both parts appear to be only in juxtaposition, so that the baskets not only always closely embrace the cell bodies, but penetrate with single processes into the granular layer. It is important further to note that the pencil-like arrangement of branches of many cells participate in the formation of one basket. Kölliker confirms Ramón y Cajal's observations that a cell only possesses one process which lends itself to the formation of a fibre basket. Besides the descending branches which go to form the basket, the nerve processes of the cells in question give off here and there towards the surface of the molecular layer processes, which behave as ordinary protoplasmic processes.

The cells which participate in the formation of the above described fibre basket, and which for simplicity's sake should be called basket cells (Korbzellen), belong, as has already been remarked, to the deeper parts of the molecular layer. The number of these elements corresponds to the number of Purkinje's cells, and is therefore sparse in the region of the furrows. Still there may be other variations in the length of their processes and in the number of their pencils. Since one cannot follow one fibre from beginning to end, it is impossible to define its actual length. In the human species the basket cells measure 11 to 20 micromillimetres, the molecular layer .15 to .14 mm., and the part of the layer containing the transverse fibres .12 to .22 mm.

Following on the description of these remarkable basket cells, their branches, and the fibre basket, the question naturally arises if they are really nerve elements, as Golgi and Ramón y Cajal believe them to be, or glia cells.[2] This is no easy matter to decide, as by Golgi's method both neuroglia and nerve cells are stained by silver. Kölliker inclines to the view of Golgi, and states the following as matter of fact:—firstly, all transitions exist between the basket cells and the small undoubted nerve cells of the molecular layer; secondly, the glia cells have, without exception, numerous branches passing off from all sides, mainly with sharpangled divisions; and, thirdly, the basket cells are, like nerve cells, stained deep black, while glia cells have a red or yellowish-brown appearance. If the basket cells are really nerve cells, they exhibit structure of quite a peculiar nature, and the relation of branches of nerve cells to other such cells as exist here are nowhere else to be seen. This is surely no reason against this interpretation, since the recent examinations upon the construction of the retina and the brain show that we have to await much that is astonishing from further researches in this sphere.

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[1] Kölliker has applied distinctive names to these structures. The basket or pencil arrangement of fibres he has called *Faserkörbe* and *Faserpinsel* respectively. The cells he has called basket cells (*Korbzellen*).

[2] In this connection it is to be remembered that Gierke speaks of the "Neuroglia holes" of Purkinje's cells, "Die Stutzsubstanz des Centralen Nervensystems," II. Theil., in *Archiv für Mikr. Anat.*, Bd. xxvi., p. 188.

4. *Purkinje's Cells.*—These are obtained readily in the human brain, and by Golgi's method present a picture of wonderful beauty. Their branches do not anastomose, and they pass out through the whole thickness of the molecular layer. Kölliker has not seen any connections between them and the pia mater or vessels, and only to a very small extent with the nerve processes of the small granular cells.

In relation to the position of Purkinje's cells, Henle first pointed out that their ramifications spread out in one single level, and were leaf-shaped, and that these leaves without exception lie in the cross plane of the convolutions. Deep tangential sections give good information upon the position of the bodies of the cells; these show that they are somewhat nearer together in the longitudinal than in the cross direction, and are not arranged in regular succession. Golgi has so well described the nerve processes of these cells, and Ramón y Cajal has confirmed it, that it is only necessary to add that Kölliker also saw the lateral branches which pass back into the molecular layer.

5. *The Nerve Fibres of the Granular and Molecular Layers.*—Of the three kinds of fibres which Ramón y Cajal describes as ramifying in the granular and molecular layers, Kölliker believes that the sub-sections (*a*) and (*b*) described above belong together, and correspond to the system of nerve-fibres which Golgi saw diffusing from the folia into the cortex. These varicose black fibres show inside the folia single acute-angled divisions, and so soon as they enter the granular layer they spread out into delicate rich ramifications, which pass through the granular layer and appear to end partly there and partly in the deeper portions of the molecular layer, *i.e.*, they end free, and are not further stained. In many preparations these endings show nothing special except slight varicosities, in others knotty or bushy swellings; it is probable that these conditions are artificial. As to Ramón y Cajal's third category, the so-called "Nidos Cerebellosos" which encircle Purkinje's cells, Kölliker only saw them

in a single preparation from a young dog, and is disinclined to pronounce any opinion on them.

6. *The Medullated Fibres of the Cerebellum.*—For the investigation of these fibres, we possess important means in the methods of Weigert and Pal.

At the borders and lateral parts of the convolutions, where the grey matter pushes against the white substance, single fibres and fibre bundles pass off from this in order to pass through the entire thickness of the rust-coloured layer, and to form in it a plexus, which takes up the granules in its meshes. In the deep parts of the furrows, different relations are found; here the white matter shows arching fibres, which, similar to those of the great brain, appear to act as commissural fibres of the convolutions, and only give off a few fibres to the rust layer. On examining this plexus more carefully, one finds that it contains a certain number of coarse fibres, which run straight towards the molecular layer, elements which undoubtedly are in great part continuations of the axis-cylinder process of Purkinje's cells. On the other hand, one cannot help thinking that the plexus of the rust layer consists entirely and alone of such fibres as Henle surmised. Still much more certain is it that it contains an excess of medullated fibres, which penetrate into the molecular layer. If the plexus is followed right up to Purkinje's cells, one finds that its elements pass under the cells into a layer of cross fibres, which, on cross section, consists of discrete bundles which are joined together by numerous fine bridges. Longitudinal sections complete the picture, and show the bundles as a not sharply-defined round collection of points. From these superficial cross bundles a quantity of oblique and straight ascending fine fibres develop, which, encircling Purkinje's cells and passing between them, enter the molecular layer, and in this strike into a longitudinal direction. In clean cross sections these elements appear as small points, which lie sometimes outside, sometimes between Purkinje's cells; and in longitudinal sections of the convolutions one finds a zone of fibrils running parallel to their surfaces. Besides these elements one finds a certain number of radial (straight and slightly oblique) ascending medullated fibrils in the molecular layer, of whose quantity it is not easy to make a calculation. They are, as a rule, few in pure cross sections, abundant on longitudinal sections of the convolutions. With reference to the diffusion of these medullated fibres throughout the molecular layer, recent examinations have given different results than previously were noted. There is no doubt that they are more numerous in the inner fourth or fifth parts of this layer, but they are also observed in the superficial strata, just under the pia mater.[1] Do the medullated fibres of the cerebellum possess branches? It is extremely difficult to define such branches, except in the molecular layer. The reason of this difficulty is found in the fact that the medullated sheath is absent at the divisions, and therefore escapes staining. This has long been known to occur at the division places of peripheral nerve fibres. Ramón y Cajal and Flechsig think that this occurs in the central nervous system. The former has seen non-medullated portions in the medullated nerve processes of Purkinje's cells, while the latter has described non-medullated places in the already medullated nerve processes of the pyramidal cells of the cortex cerebri, where they divide and give off lateral branches. Kölliker has also seen such conditions in the fibres of the granular and molecular layers, but could not say whether they were natural or only imperfectly stained. In well-stained Weigert preparations from man and cats he saw such divisions in the molecular layer in only thirteen cases.

7. *The Neuroglia.*—In the white matter numerous large star-shaped cells are found, whose long-branched processes course between the nerve fibres, and apply themselves to the vessel walls. Similar cells are seen in the granular layer and at the border of the molecular layer, which send their long-branched processes right through this layer to its surface, to end at the inner aspect of the pia mater in a small club-shaped swelling.

C. THE CEREBRAL CORTEX.

Ramón y Cajal has examined the cortex in small mammals (rats, rabbits, &c.). He divides it into four layers. *First or molecular layer.*—This, the most superficial layer, is composed of nerve fibres, nerve cells, and the protoplasmic expansions of the subjacent pyramidal cells. The nerve fibres are extraordinarily abundant, and run parallel to the surface of the cortex; some of them have been observed to pass into the subjacent layers, and become continuous with axis-cylinder processes. Of the nerve cells three kinds have been described:—(*a*) Polygonal cells, not numerous, and possessing protoplasmic processes which pass off in all directions. (*b*) Fusiform cells; these he found lying in a horizonital antero-posterior direction. They have only two protoplasmic processes, one from either pole. Owing to their great length it is almost impossible to follow out their whole course, but they bend and end free under the surface of the cortex. The axis-cylinder processes (for there may be two or three of them) arise from the protoplasmic expansions at a considerable distance from the body of the cell. They give off at right angles numbers of ascending collaterals, which end free in this layer. It is not uncommon to find a supernumerary axis-cylinder. The fusiform cells are not numerous. (*c*) Triangular cells; these are much more plentiful than the preceding; of the three branches which each cell gives off, one is descending and bifurcates. There are two, three, or more axis-cylinder prolongations, which always arise from the protoplasmic processes. Some pass to the surface of the layer, and others terminate free. The protoplasmic expansions of subjacent pyramidal cells ramify to a considerable extent in this layer, and end under the free surface. There are also oblique and arciform branches which end in different parts of this layer. Here, then, is an arrangement similar in all respects to that which obtains in the molecular layer of the cerebellum. In his previous works Ramón y Cajal had considered the protoplasmic processes not as absorbers of nutrition, but as the means of communication of nervous action between neighbouring cells and elements at a distance. In applying this principle to the molecular layer of the cerebral cortex, one observes that the fusiform and triangular elements, by means of their multiple axis-cylinder prolongations and very long protoplasmic expansions, are in contact with and intermix with a great number of the terminal protoplasmic processes of the subjacent pyramidal cells. It is not unlikely that some of these may functionate by contact. It is on this supposition that the term "association cells" has been given to the elements of the molecular layer.

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[1] It seems probable that in Weigert preparations dark-stained glia fibres may be mistaken for very fine medullated nerve fibres. The former are of equal diameter and always serpentine; the latter are straight and usually slightly varicose.

Second layer (layer of small pyramidal cells).—In mammals these cells are not proper pyramids, but are polygonal or star-shaped. The protoplasmic processes pass off from the upper and lateral angles, and terminate in the molecular zone. The axis-cylinder descends, but its termination is yet the subject of much investigation. The author states that he has traced it into the white matter, and onwards into the corpus striatum (from cells in the frontal lobes), and into the corpus callosum (from cells in the Rolandic area). In their course they give off collaterals.

Third layer (layer of large pyramidal cells).—This layer passes insensibly out of the previous one. In mammals it resembles that of man, except that the cell bodies are less pyramidal and more ovoid, and the protoplasmic processes are shorter. The axis-cylinder descends in a straight line into the white matter, where it becomes continuous with an association or projection fibre. It gives off many collateral branches, which assume a horizontal and oblique course, and end near the molecular layer. Nearly all the collaterals end in a small expansion without any branching.

Fourth layer (layer of polymorphic cells.)—The cells are mainly globular, but some are fusiform, and of various pyramidal types. They all have ascending and descending protoplasmic processes, the former of which end in the middle layer of the cortex, the latter in the white matter. The greater number of axis-cylinder expansions descend and give off fine collateral branches, which end as a delicate fibril in the white substance. This layer is in contact with the white fibres.

Scattered throughout these layers are certain other elements which require description:—1. *The sensory cells of Golgi.*—They are met with in all the layers, but mainly in the fourth or deepest zone. Their protoplasmic processes pass off in all directions, and are of considerable length, but they do not reach the molecular layer. The axis-cylinder descends, has a short course, and ends free in a complex ramification. Ramón y Cajal does not hesitate to disprove their sensory nature, as advocated by Golgi; he inclines to the view that they are "association cells." 2. *Cells with an ascending axis-cylinder.*—They are triangular, and lie amidst the small pyramidal cells. The axis-cylinder enters the molecular layer. Here it divides into horizontal fibres of great length. But all the cells are not prolonged so far as this layer; a similar disposition of branches takes place also in the second and third layers. 3. *White fibres which ramify in the grey matter.*—These are amongst the largest fibres which cross the cortex. They are larger than the axis-cylinder process of the large pyramidal cells. They penetrate the cortex at a right or obtuse angle, and divide into two or three large branches. These repeatedly divide, and end in the layers of small and large pyramidal cells. These fibres never anastomose with other fibres in the grey matter, nor do they terminate in cells. Ramón cannot confirm Golgi's belief that they are sensory. 4. *The nerve plexus of the grey matter.*—The intervals between the nerve cells are filled in by varicose fibres, formed by collaterals of the pyramidal and polymorphic cells, by the terminations of the sensory cells of Golgi, by the collaterals and terminals of the cells having an ascending axis-cylinder, by the collaterals of the association fibres of the white matter, and by the collaterals and terminals of the fibres coming from distant central organs, and from the corpus callosum! The enormous complexity of the nerve network of the grey matter is sufficiently apparent from this description. No idea of the innumerable ramifying fibres in the cortex can be obtained by the Weigert-Pal method.

The white matter. 1. *Fibres proper to the cortex*, or association fibres.—The axis-cylinders of the cortical cells are continued into these fibres, either by simple bending at a right angle, or by dividing into a T or Y in the white matter; the branches of bifurcation pass in opposite directions. After some distance the fibres turn back into the cortex, and end by dividing. In the outer part of the hemispheres the fibres change their direction and form a large transverse descending tract—the fasciculus transversalis. When the fibres above mentioned divide into a T or Y, one of the branches goes to the corpus callosum. The association fibres give off collaterals, which pass at a right angle into the cortex, behaving just as the collaterals of the column fibres of the spinal cord. The presence of collaterals of the white fibres of the brain is now a well-known fact. 2. *The corpus callosum.*—The fibres of this structure have a horizontal course in the middle portion, but laterally they are ascending. They maintain their parallelism until they reach the subcortical white matter, where they bend amongst the arcuate fibres, and pass into the grey substance. They give off collaterals, which also pass into the cortex. It is not yet certain how these fibres commence or terminate. Some appear to be direct continuations of axis-cylinder processes; others are collaterals of the axis-cylinders of projection and association fibres. Their terminations are probably delicate ascending fibrillæ, which are found distributed through the middle and lower layers of the cortex. 3. *Projection fibres.*—These are the direct continuations of the pyramidal and polymorphic elements of the cortex. They give off a great number of collateral branches. They pass into the corpus striatum, where many are seen to give off very fine ramifying branches, which end in the cells of this ganglion.

Some general conclusions are worthy of note. One cannot tell by any special morphological character cells whose axis-cylinder constitutes either an association, a projection, or a commissural fibre. No direct anastomoses are observed between the nerve fibres of the grey matter. Transmission of nerve impulses must therefore be carried out through contact. The human cerebrum owes its superior activity not only to the great number of its cells, but to the extraordinary richness of its association paths —*i.e.*, the collaterals, axis-cylinder, and protoplasmic expansions.

Van Gehuchten describes the *granular layer of the cerebellum* as composed of three kinds of cells—small and large nerve cells and neuroglia cells. Each small cell has a polygonal body, from each angle of which there passes a protoplasmic process, which ends freely in a tuft. The axis-cylinder rises from a protoplasmic process, and passes towards the molecular layer, where it bifurcates; its divisions end free. The large cells are met with in close relation to the cells of Purkinje. They have many protoplasmic processes, some of which pass to the surface of the cerebellum. Others are horizontal, and end in the granular layer. The *molecular layer* is rich in small cells. They give off protoplasmic processes, which pass upwards and downwards, while the axis-cylinder expansion assumes a horizontal course. Some of the collaterals of this pass to Purkinje's cells, which they envelop in a tuft, the so-called *Korbzellen* or basket cells of Kölliker. The protoplasmic processes of *Purkinje's cells* extend into the molecular layer; the axis-cylinder courses through the granular layer to the white matter.

Further study of the cerebellum has given rise to the same general conclusions regarding it as in both the cerebrum and the spinal cord. The true nerve element is the cell with its prolongations. The nerve elements are independent of each other, the protoplasmic processes end free, and, whatever its disposition, so does the axis-cylinder. Finally, the transmission of impressions here, as throughout the cerebro-spinal system, takes place not by continuity of structure, but by contiguity or contact.

Van Gehuchten has also paid attention to certain doubtful points in the spinal cords of small mammals. Firstly, the relation of the anterior nerve roots to the cells of the anterior horn. He finds that each anterior nerve root is the axis-cylinder prolongation of a cell. The protoplasmic processes of these "root cells" are numerous, and form an inner group, which passes by the anterior commissure to the opposite anterior horn; an antero-external group which passes to the periphery of the grey matter; and an antero-posterior group which passes into the grey matter.

Gehuchten's researches upon the posterior roots support the statement that some of these root fibres have their origin in cells in the grey matter of the cord. This, we have seen, is opposed to the views of Golgi, Ramón y Cajal, and Kölliker. The great majority arise from cells in the posterior ganglia, and divide into ascending and descending branches. Both give off collaterals, which pass into the grey matter. A great many of these branches end in close relation to the cells of Clarke's column. Many of the fibres themselves end free in the grey matter at all levels of the cord, but others terminate in the nuclei of Goll and Burdach. The longitudinal fibres of the antero-lateral column behave just as those of the posterior—*i.e.*, that at variable distances they give off collateral branches, which pass into the grey matter and end free. All the nerve fibrillæ in the grey matter form a complex network. They remain quite independent of each other. Collaterals of the lateral column fibres have been seen to cross by the posterior commissure, and end in the grey matter of the opposite posterior horn.

The study of the structure of the spinal cord confirms what Golgi originally noted—that the cells do *not* anastomose through their protoplasmic processes. Each cell with its processes forms an independent autonomous structure. His has shown from his embryological studies that every nerve cell, with its processes, is formed by the transformation of a neuroblast. Nerve cells vary immensely in size, form, and richness of processes; but there is one constant structure, the axis-cylinder prolongation. It arises directly from the body of a cell, or from one of the expansions. It is distinguished by the cleanness of its contours and its regular course. It retains an unaltered diameter for a long distance; it bifurcates and gives off collaterals. It terminates blindly in one, two, or more branchlets. One can therefore divide these elements into two main sets—one occupying the top of the cerebro-spinal axis, whose process conducts centrifugally; and the other the lower part, whose process conducts centripetally. But an intermediate class exists, whose axis-cylinder process bifurcates and conducts upwards and downwards ("mixed cells"). An important fact in nerve physiology is therefore confirmed—that the axis-cylinder process of a nerve cell is a conductor, conveying any form of impression, and transmitting it, by collaterals or terminals, to corresponding structures with which it comes in contact.

Dr. Luigi Sala deals with the *minute anatomy of the pes hippocampi* elucidated by Golgi's method. The method shows that the structure of this portion of the brain is not so complicated as one would imagine from the descriptions of Kupffer, Meynert, Huguenin, Krause, and others.

Golgi divides the pes hippocampi into the four following layers:—

1. The inner or first layer of nerve fibres (alveus).
2. The grey convoluted layer, or layer of large ganglion cells.
3. The outer or second layer of nerve fibres (lamina nuclearis).
4. The layer of small ganglion cells (fascia dentata). Sala's description is based upon these four layers.

1. *The Alveus.*—This consists of a layer of thin medullated nerve-fibres running parallel to each other, and forming a bundle which covers the whole cornu ammonis. The fibres arise from the large cells of the second layer, from the small cells of the fascia dentata, and from the cells of the gyrus hippocampi. Numerous glia cells are scattered among the fibres, and here, as elsewhere, they are attached by their processes to the walls of the blood-vessels. In addition, a few nerve-cells have been found, whose axis-cylinder process finds its way into the subjacent grey layer. Golgi believed that these cells partly belonged to the grey layer. The alveus is in relation on its free surface with the cells of the ependyma ventriculorum. The bodies of these cells are continued into the alveus by a long process, which, contrary to previous belief, divides shortly after its origin into numerous branches that are attached to the blood-vessel walls and to the glia cells already mentioned, while others lose themselves in the nerve substance, apparently passing as far as the large pyramidal cell layer.

2. *The Grey Convoluted Layer.*—In addition to the characteristic cells of this layer, numerous medullated nerve-fibres are present. They consist of fibres which arise from the lamina nuclearis, and are in transit through this layer on their way to the alveus and the fimbria, and partly also of fibres which pass from the fascia dentata to the alveus. Medullated fibres are also present where this layer bounds the lamina nuclearis. The cells of the layer are pyramidal, and are continued inwards by a protoplasmic process which, dividing and subdividing, ends at the numerous glia cells scattered throughout the layer, but especially in its deeper parts near the lamina nuclearis. Filaments from these processes pass through the alveus to fuse with the cells of the ependyma. The cells occupy one or more rows near the outer limit of the layer. The nerve-process passes off from the side next the alveus into which it goes; a short distance from the body it gives off filaments, which form a delicate network in the grey layer. From this there can be traced many fibrils which go on the one hand into the alveus, on the other into the next layer.

3. *Lamina Nuclearis.*—This consists of medullated nerve-fibres arising similarly to those of the alveus. Running parallel

to the grey layer, they turn backwards with this layer and are continued behind into the white matter, which covers the subiculum and gyrus hippocampi.

4. *Fascia Dentata.*—Of this there are two layers, which must be separately considered—a superficial, formed of medullated nerve-fibres, and a deep one, composed of the characteristic cells and their processes.

(*a*) The superficial white layer of the fascia dentata consists of fibres similar to those of the alveus and the lamina nuclearis. It commences in a series of medullated fibrils, running together to form a bundle, which is most evident where the fascia dentata is folded backwards; further on it is lost in the superficial white bundle of the lamina nuclearis. Although closely associated, these two layers can be separated by the presence of numerous blood-vessels which lie between them. The fibres arise mainly from the small spherical cells of the region, and to a less degree from the large pyramidal cells.

In the part of the fascia dentata which remains uncovered, one readily sees that the superficial white layer is not at the periphery of the fascia itself, but that between this and the bundle there is a thin layer which contains no nerve-fibres. This is at the place where the fascia is in relation with the subiculum cornu ammonis. Golgi's method shows that this area is very rich in neuroglia cells, in which many of the protoplasmic processes of the small spherical cells end. It contains also a few nerve-cells, whose nerve-processes are lost in numerous fine branches.

(*b*) The characteristic cells of the fascia dentata are small and spherical, or oval in shape. They lie in the deeper parts of the layer, where they usually form a single row. They present a great likeness to Purkinje's cells—*i.e.*, from one side there spring the protoplasmic processes, from the other the single nerve-process. The former, five or six in number, branch dichotomously, and passing through the white layer, reach the neuroglia cells already described. The nerve-fibre process gives off lateral branches, which in their turn divide, and so come to form a delicate network which exists throughout the whole course of the fascia dentata. From this network numerous secondary processes arise, which are continued as nerve-fibres (type 2), but the axis-cylinder process can also be traced in many cases through the network without losing its individuality (type 1). From this network these fibres pass away to take part in the formation of the alveus and the fimbria, and to a less degree in the lamina nuclearis and superficial white layer. As long as the fibres are in the network they remain non-medullated, and possess peculiar triangular and oval swellings.

In the network a considerable number of neuroglia cells are found, as well as isolated nerve-cells. These latter are present mainly in the outer peripheral part of the net, and possess protoplasmic and nerve processes. It is probable that these cells belong to the terminal portion of the grey convoluted layer.

1. *Some General Facts brought out by Golgi's Method.*—Of the many processes which a nerve-cell gives off, only one—the axis-cylinder process—is in direct relation with a nerve-fibre; the others—protoplasmic processes—mainly subserve the nutrition of the nerve elements through a direct relationship with neuroglia cells and blood-vessels.

2. The axis-cylinder process is related to nerve-fibres in one of two ways:—(*a*) In the first, or motor type, this process passes directly into a nerve-fibre in spite of lateral ramification. (*b*) In the second or sensory type, a network of nerve-fibrils intervenes between the cell and the nerve-fibre.

3. The protoplasmic processes are neither continuous with nerve-fibres nor anastomose with them. They probably functionate by contact with other such processes, and so conduct from cell to cell. The following facts support the belief that the protoplasmic processes are essentially of a "nervous" nature (Kölliker). (*a*) In structure and chemical constitution they are similar to the axis-cylinder process of nerve-cells. (*b*) It is not uncommon for a nerve-process to arise from protoplasmic processes.

4. *Continuity of structure is not essential for the propagation of nerve impulses; cells and fibres may functionate by contact only.*—This is probably the most important fact yet pointed out from the study of the nerve elements by this method. Kölliker has shown that this occurs in the following situations:—(*a*) In the sensory root fibres, which end free in the grey matter of the cord and medulla. (*b*) At the terminations of the lateral branches of the nerve process of many of the cells of the grey matter. (*c*) At the terminations of the longitudinal fibres and collaterals of the anterior and lateral pyramidal tracts in the grey matter of the anterior horns.

It is, therefore, apparent from these observations that the three most important functions of the spinal cord, the propagation of motor, sensory, and reflex impressions, takes place not by a direct continuation of nerve structures, but by mere contact of nerve-cells, nerve-fibres, and their processes.

WILLIAM ALDREN TURNER.

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Reviews and Notices of Books.

A Dictionary of Psychological Medicine, giving the definition, etymology and synonyms of the terms used in medical psychology with the symptoms, treatment, and pathology of insanity and the law of lunacy in Great Britain and Ireland, edited by Dr. HACK TUKE, M.D., LL.D. 2 vols., 8vo. 1477 pp. London, Churchill, 1892.

Dr. Hack Tuke has just brought to a successful issue a great enterprise which does the highest credit to English medical and psychological science. He has brought together in the form of a dictionary all the facts and ideas necessary to the scientific study, as well as to the practice, of mental medicine. Such a work could not come in better time, for at present these subjects, formerly somewhat neglected or despised in literature and science, are attracting the attention of all. It is no longer possible to deal with philosophical, medical, legal, and even literary subjects without paying regard to the researches of scientific psychologists of all countries. It will be a great help to find in the convenient form of a dictionary numerous references otherwise difficult to get at.

No one was better qualified for the undertaking than Dr. Tuke; his well-known writings on psychiatrics, his work on "Mind and Body," which already contained the germ of more recent researches gave him the necessary authority to lead the numerous and indispensable collaborators. I may be allowed to add that his simple and cordial *amabilité*, the affectionate feelings which he naturally inspires to all those who have known him, have allowed to bring together around him a large number of writers very different from one another.

These collaborators are indeed numerous: 128, and of all countries. Of them 84 are British, 17 French, 12 German, the others American, Dutch, Swiss, Italian, Austrian, Belgian, and Russian. Hence we may say that the dictionary is not a purely English work, but that it is a resultant of thought in different counties. Among the chief authors, we must mention Dr.

Urquhart, who presided over the bibliographical part, Drs. Pietersen and Willoughby, who drew up the short definitions of terms and the etymologies, met with in such large numbers. These data form one of the most useful parts of the book, and give a multitude of precise indications to the searcher. Perhaps I may here express a regret: Would it have been impossible to indicate by the side of every new and rare term, the name of the author who first propounded it, and the place where he did so?

The information given in the body of the work is completed by tables placed at the end of the second volume, by lists of the leading psychological journals and societies in different countries, by an index of legal abbreviations, etc. In a word, one is able to find immediately the practical data, without which it is at the present time so difficult to treat a scientific question.

The dictionary is moreover a complete treatise of psychiatrics, and the chief articles it contains may be grouped under five heads: 1, Historical; 2, Pathological and descriptive; 3, Anatomical and Physiological; 4, Psychological; 5, Legislative.

1. *History of Mental Medicine.*—The scientific study of lunatics appears to have a recent origin; we are too much inclined to believe that it began in the last century. It is not superfluous to remark therefore that in early times the examination of such cases had been undertaken and that some old authors have come near our most recent theories. Dr. Tuke presents these historical researches in an important article which prefaces his first volume. We are made to witness the evolution of lunacy through the centuries; there always was madness among men, because the organ of thought has always been exposed to numerous perturbing influences. There are scanty, but clear, documents concerning insanity among the Egyptians, the Persians, the Hebrews, the Greeks and the Romans. The madness of Hercules, as given by Appollodorus, is an accurate description of a typical case of epileptic delirium. Hippocrates, in the fifth century B.C., spoke of lunatics in a remarkable manner, and knew well how to connect their symptoms to cerebral disturbances. Asclepiades, Themiston, Celsus do not understand their cases so well; the latter advises brutal methods of treatment, too often followed since. Cœlius Aurelianus denounces these useless and culpable violences. "Here was a physician far in advance of all who had preceded him, and the marvel is that he should have been equally in advance of almost all who succeeded him. We desire to do him all honour and to render him the homage which is so justly his due by placing him on the

highest pedestal in the temple of fame amongst the physicians of antiquity so far as related to the medical and moral treatment of the insane."

Let us mention, in the middle ages, Paracelsus, Felix Plater, Sennert; in the 18th century, Prochaska Sydenham, and Willis, a clergyman, who undertook the cure of King George III.; we then read the epoch which Dr. Tuke hails as a new era, when Pinel at Bicêtre, and William Tuke at York Retreat, brought about a thorough revolution in the treatment of the insane.

Dr. Tuke breaks off his history of insanity at that period; but this history is supplemented by a series of papers on the organisation of asylums, and on the legislation of the insane in the principal countries.

The inquiries concerning Germany, America, Great Britain, Scotland, Spain, France, the colony of Gheel, Holland, Italy, Russia, Scandinavia, Switzerland, Turkey, brought to light many valuable documents. But it appears to us that these articles rather lose sight of medical studies.

We would have been interested in following the history of these studies in England after the time of William Tuke, and in France after those of Pinel and Esquirol; Moreau Morel, and Baillarger, a generation more engrossed, perhaps, by psychological questions, than our contemporaries. It would also have been interesting to briefly recapitulate the history of the spiritualistic school of Germany, of Stahl, Heimroth, Ideler, etc. These expressions of regret are prompted by the lively interest which Dr. Tuke's historical sketches have had for us, and by our wish to see him expanding this history of insanity.

2. *Mental Pathology.*—The first, and perhaps the most intricate, of the questions which we meet in our investigations of insanity is that of the classification of the various mental maladies. None of these classifications is perfect, and it is probable that none of them will be long-lived, but it is at least useful to know the chief ones.

Dr. Tuke, in a paper dealing with the subject, points out the classifications which are best known, or at least, most usual, in England. It would perhaps have been as well to indicate, and to more fully discuss, some rather curious classifications adopted by modern German and French authors.

Another paper deals with the diagnosis of mental diseases, and the psychological examination to which a patient should be subjected; this examination is rather vaguely defined, perhaps for the reason that even the normal psychological functions are not defined with sufficient precision.

Some of the descriptions of mental diseases have been elaborated with great care. The definition of these, often a difficult task, is summed up with great precision, the symptoms are described in an abridged form, but nevertheless in a manner sufficient for the purpose; the diagnosis, the prognosis, the pathological anatomy, and the treatment of each disease occupy a special paragraph; for instance, melancholia, and mania under all forms, are described in several articles.

We do not entirely agree with the author as regards the prognosis he gives of hysterical mania; it is customary, in medical language, to assign a very favourable sense to the term "hysterical," and to make any mental aberration that may be called "hysterical," appear as of a benign nature. This is far from being always correct; the hysteria which tends to assume the form of a characterised psychosis, is, in most cases, very serious. The first attack of hysterical mania may possibly pass away, leaving behind nothing worse than great mental debility. But it usually reappears ere long and according to Prof. Falret, whose opinion based upon ample observation, hysterical mania is one of the chief causes from which precocious dementia arises.

Dementia, which occupies so important a place in the history of mental diseases, is analysed with full detail by Dr. T. C. Shaw; paralytic dementia or general progressive paralysis is discussed by Dr. T. Mickle, who not only sums up all our present knowledge, but carefully points out all problems to be still elucidated.

Dr. Tuke endeavours to explain the somewhat obscure meaning of the word, "paranoia," and Dr. V. Parant presents a general outline of the delirium of persecution. We now reach those interesting forms of insanity where the mind does not seem to be entirely diseased, where the patient retains at least a partial consciousness of his condition, and where only a restricted portion of his thoughts seems to be affected; it is somewhat difficult to consider such maladies as insanity, properly speaking, and one is inclined to dissimulate them under such terms as neurosis, emotional neurosis, neurasthenia, or hysteria. We should not be misled by these appellations, for we have to deal with real diseases of the whole mind when we meet these forms of insanity. The various monomanias which play so conspicuous a part in the psychoses, are all separately described according to their importance.

There are interesting articles on pyromania, dipsomania, morphiomania, suicidal mania, homicidal monomania, which is well defined in the following terms: "It is a craving for the commission of homicide, without any intellectual or passional derangement and with persistent consciousness, and anxious struggle against the besetting impulse, and, finally, after committing the deed, a lasting and consecutive moral depression." Contagious or communicated insanity, moral insanity, the insanity of denial, the delirium of doubt, may likewise be annexed to this group. The greater part of these forms, which are handled by Dr. Legrain and other authors, are presented as symptoms forming part of one great morbid type, that of hereditary insanity, and bear the title of Stigmata of mental degenerates.

As a matter of fact this theory has been for some years past expounded in various countries, and principally in France, by Prof. Magnen and his school; but, nowadays, this way of representing matters has been pretty sharply criticised. I regret that in the dictionary there are not found sufficient evidences of those discussions, which ought to have been taken into consideration.

There is evidently something very rational in this theory. "These patients," says Dr. Legrain, "retain consciousness, but they show a fundamental derangement of the mind by which the impulse is accounted for." Doubtless, the fixed idea, like the suggestion to which it is near akin, is not a primitive phenomenon. I mean that it cannot be the *first* pathological phenomenon, and that the fixed idea cannot grow up in a mind wholly sound in all other respects. The fixed idea is a kind of parasite which develops itself in a complete and isolated manner, apart from the mass of other ideas, and, as it were, independently of personal consciousness. In order to render a similar development possible, it is necessary that there should be a special state of mind allowing such an isolation, such a separation of thought; and the theory of degeneracy seems to us to be incontrovertible on this point.

But let us now examine the nature of the mental perversion which admits of suggestion and a fixed idea. Will it be sufficient to simply assert that it *is* a degeneracy, and that it depends upon heredity, which, like a *deus ex machina*, steps in and explains everything? I believe that this expression, if not absolutely incorrect, is at least far from being precise. In the first instance, heredity intervenes in nearly all mental disorders, almost without exception; it produces idiots, epileptics and the victims of typical persecutional hallucinations.

It must be admitted that the latter have insanity among their ancestors, and that they display the stigmata of degeneracy just as much as the victims of obsessions and doubts. Why should this influence of hereditary degeneracy be confined to a special category of patients? On the other hand, this weakening of mind which makes fixed ideas possible may be due to many other causes besides heredity properly so called, *e.g.*, contagious maladies, such as typhoid fever, organic debility, starvation, overwork, emotional strain. The patients fail to properly concentrate or synthetise their thoughts; they become susceptible of suggestions and predisposed to fixed ideas for a period of more or less duration. This is especially the case with the "neurotic" state. It is daily becoming more and more recognised that neuroses, neurasthenia, and hysteria, are the real soil upon which doubts, interrogations, obsessions and impulses, take root and grow. Doubt exists with the majority of such patients, generally bearing upon all sensations, and upon all images which are badly perceived and synthetised in the mind. This doubt may easily become fixed upon some particular point, it may assume the shape of obsessive interrogations, and develop into a downright delirium.

Are we then to designate all these mental disorders as degeneracies? Yes, possibly, in one sense; for it is evident that a neurasthenic patient has not the mental power which he ought to have, and that he is in a fallen degenerate state. But then the word loses all its precision, for it thus becomes applicable to almost all maladies without exception.

I do not intend to discuss here this problem of classification; but simply wish to point out its difficulties. It matters little, in reality, what designation we use; and it is sufficient to establish the fact, as has been done by Dr. Legrain, that fixed ideas originate in a predisposition of the mind, and to carefully analyse that mental condition.

Dr. Tuke is so thoroughly convinced of this necessity, that he does not hesitate to devote several exhaustive paragraphs to the study of neuroses. Not long ago, hysteria was described along with the organic disorders of the cord, disseminated sclerosis, and tabes, but it is far more correct to classify it among the psychoses. I even believe that it is destined to play a very important part in works on mental medicine; for it is simply an insanity the phenomena of which are accessible to direct observation and analysis and even to a process of experimentation.

We must therefore accurately ascertain the mental condition of hysterical patients, before entering upon the study of more complicated forms of insanity.

The description of hysteria is given from a clinical standpoint in an elaborate article by Profs. Charcot and Marie. I do not think that it would be right to look upon it as giving the French theory on hysteria. It is not, in fact, a theory; but rather a description of the attacks, of anæsthesiæ of contractures, such as are met with everywhere, even in England. Nevertheless, we have been greatly interested in another paper on hysteria by Dr. H. B. Donkin, which, we are told, represents the opinion of the medical faculty in England regarding this neurosis. The clinical description differs very little from the preceding one; Dr. Donkin lays less stress upon the attack, the serious nature of which is rather lightly touched upon, but, greatly to our satisfaction, he insists rather more upon the psychological interpretation of hysteria. "There is much less risk of error in roughly classing hysteria as a species of insanity than in taking one of its physical expressions, the hysterical fit, for instance, or sexual disorder, as its central fact. . . . One French school of neurologists, who have worked so exhaustively and minutely in this field, have of late espoused the long prevalent English view of the *rationale* of these phenomena, some of its most modern opponents ably illustrating many hysterical phenomena by the facts of hypnotism, and indeed claiming *suggestibilité* as the essence of the hysterical neurosis."

I should have been glad to know the English authors who have clearly demonstrated the psychical nature of hysterical accidents, prior to the book of Briquet, who considers hysteria as an emotional malady, and to the teachings of Charcot concerning traumatic paralysis; but I am happy to learn that this opinion has so long been held in England.

Further on Dr. Donkin has, it seems to us, somewhat lost sight of the method which he had so correctly indicated at the beginning. No doubt he distinctly recalls the theory of psychical paralysis "dependent on idea." He also points out the lack of effort, the minimised will-power of the patient who does not even endeavour to move, but he scarcely gives us a psychological explanation of the other phenomena, of the anæsthesias, contractures, convulsive attacks, etc. The disturbances of memory, of volition, of attention, and of emotion might be indicated with greater precision; but these slight wants are of minor importance, and we do not dwell upon them, since, on the whole, the conception of hysteria seems to us to be very accurate.

Our thanks are due to Dr. Donkin for the clearness with which he expresses himself concerning somnambulists and the mediums: "It is among hysterical sufferers that many instances of natural somnambulism and most of the subjects of induced hypnotism are found. Notwithstanding the recent teaching that all human beings are potentially hypnotisable, it is certain, from general experience, that human beings are hypnotisable in direct proportion to their nervous instability." These are words which it would be well to repeat in England, where so many authors still represent somnambulism and automatic writing as being a normal characteristic of people of sound mind.

We pass over numerous articles on the mental and nervous pathology to call attention to a paper on neurasthenia, and a series of fine essays on the real degenerates, imbeciles, idiots, etc., considered from a physical and moral standpoint.

The reading of all these articles gives, if not a complete knowledge of, at least a clear insight into, mental pathology and the controversies raised by the investigation of the subject.

3. *Anatomy, Physiology, General Medicine.*—Although we are yet far from understanding fully mental diseases, we are at least able, nowadays, to study them by different methods. Anatomy, physiology and general medicine, each furnish facts and theories which enable us to arrive at an improved interpretation of certain forms of insanity. We note in the dictionary the articles by Prof. Benedikt on cerebral craniometry, the description of cranial modifications observed in micro-cephalic cases, the very useful information about anatomical preparations and microscopical examination of nervous tissues. The anatomy of the brain, of its membranes and its vessels, is described with great precision by Dr. A. Bruce, and forms the subject of a new supplementary paragraph at the end of the second volume.

A question of importance in our days, the morphology of cerebral convolutions, is treated in an elaborate article by Dr. Hack Tuke; several accurate figures accompany the description. We also find information relating to the specific weight of the brain, to its chemical constitution, and to its various modifications in insanity.

Passing from anatomy to physiology, we are met by the first and most important of all the problems, that is, the formation of the new being whether in health or disease; this formation depends upon the healthy or sickly disposition of the parents, upon heredity in short; the study of Heredity, by Dr. C. Mercier, is embodied in one of the finest articles of the dictionary. Its nature is not discussed (the theories of Weissmann are scarcely touched upon), but it is investigated from a practical point of view in its relations to mental aberration. Some of the most of natural somnambulism and most of the subjects of induced hypnotism are found. Notwithstanding the recent teaching that all human beings are potentially hypnotisable, it is certain, from general experience, that human beings are hypnotisable in direct proportion to their nervous instability." These are words which it would be well to repeat in England, where so many authors still represent somnambulism and automatic writing as being a normal characteristic of people of sound mind.

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Not only are neuroses considered as causes of insanity, but some of the authors, I think quite correctly look upon them as insanity itself. All these different essays tend towards the same point, viz.: the analysis of mental disturbances and of their physiological conditions.

4. *Psychology.*—Mental diseases are more complex than others and cannot be studied in the same simple manner as maladies of the body; they present two different aspects, the one is external or physical, the other is internal or psychological; it is not sufficient to describe an insane patient externally, and to demonstrate the modifications of the pulse or the urine, we must penetrate the depths of his mind and must picture to ourselves, as much as possible what he feels and what he thinks; psychological research is absolutely indispensable to the specialist in insanity; the specialists in lunacy of the time of Esquirol, of Moreau (de Tours), and of Griesinger were quite conscious of this fact; but for some time the writers who succeeded seemed to neglect those studies, and we are glad to note that these researches are now being resumed in every quarter. Is not the number and the intrinsic merit of the paragraphs devoted to pure psychology in a dictionary of mental medicine, a sign of this interesting movement, of this return to mental studies by physicians for the insane? At the beginning of the work, as a preface which was considered necessary, there will be found a long article by Mr. Coupland upon the "Philosophy of Mind"; if, on the one hand, we regret to find that this work

has not a more "medical" tendency, and that it does not lay greater stress upon pathology, on the other, we are glad to see that that the profession in England attach great importance to purely theoretical psychology. In the body of the dictionary itself, we meet with numerous articles upon the same subject, "Psychological Methods," by Dr. H. Donaldson, A. D. Waller, W. Bevan Lewis; "Psychosis," by Dr. Francis Warner; "Consciousness," by Dr. C. Mercier; "Reaction-time," by Prof. Joseph Jastrow; "Memory," by Drs. Bleuler, Ribot and Willoughby; "Attention," by Prof. James Sully; "Dreams and Hallucinations," by Dr. Hack Tuke; "Instinct," by Prof. Romanes; "Actions and Behaviour of the Insane," by Dr. C. Mercier; "Hypnotism," by Drs. Charcot, Gilles de la Tourette, Myers, etc., etc.

To review in detail all these articles would be beyond our limits, we can only venture upon a few remarks regarding some of them in order to show how much importance we ascribe to them. Let us first of all point out the principal attempts at psychology which are anterior to our epoch. Mr. Coupland justly thinks that the history of a science is conducive to the knowledge of the science itself, and he gives us some interesting information concerning Aristotle, Locke, Hartley, James Mill, etc. I hope I may be excused for passing a slight criticism, or rather for putting a simple question. Does the author seriously believe that psychology has been studied in Greece and in England only? Does he think that psychological study has descended direct from Aristotle to Locke and Hartley? Have such men as Descartes, Malebranche or Leibnitz never existed? Here we have, I think, a very serious historical question, for psychological physiology, as understood nowadays, proceed far more directly from the Cartesians than from Locke. The influence of the French philosophers of the seventeenth century was considerable, and it can easily be shown that the English psychologists, as, for instance, Dr. Hume, of whom Mr. Coupland does not make sufficient mention, have been inspired by the works of that school. Nor do I believe that German psychology begins with Herbart, whom Mr. Coupland very tardily mentions after the whole development of English psychology; it is considerably anterior and dates back as far as Leibnitz, whose theories, even in our days, play so important a part in psychological pathology.

Mr. Coupland is, no doubt, of the same opinion as myself, but perhaps he intended to present only a brief summary, and will, at any rate, excuse my criticism.

It is interesting to observe that all authors who nowadays devote themselves to physiological psychology are nearly agreed upon one general idea. The old theories upon the nature of psychical facts, upon their connection with the organism, the old metaphysical theories concerning spiritualism or materialism, properly so-called, are no longer held. The doctrine of correspondence, of concomitance between psychological and physiological phenomena, as Dr. H. Jackson observes, is sufficient for the present purposes of science. "There are," as Mr. Coupland pithily remarks, "either two sets of phenomena, heterogenous but concomitant, or one phenomenon describable in two forms of language according as it is regarded as strictly limited to one individual, or as capable of becoming a portion of universal experience." This conception seems to us to be correct; sufficient, at least, to allow experimental researches, without unduly embarassing us by metaphysical problems. But it should be properly understood, and we should accurately guage the depths into which it may lead us; if the physical and mental phenomena form two series of parallel facts, it will be necessary to study each series separately with the process adapted to it; we must study the former by physiological methods, and must endeavour to reach the psychological facts by introspection, by observation of actions and language, by time measurement, &c. It is only after a complete and independent investigation that these two series of facts should be brought into juxtaposition. A physiological fact which has been clearly demonstrated by suitable methods, may then be correctly and usefully collated with a psychological fact which has been equally well proved by the proper process. If there is an error, which seems intolerable to us, it is to invent, to construct from imagination either of these two facts and to bring it into juxtaposition with the other. Some authors delight in explaining a moral fact by imagining some physiological process or other, which they then collate with a psychical phenomenon. Thus, in the case of an association of ideas, or other very definite psychological fact, instead of accurately describing its exact nature they speak of an intercommunication between nervous fibres, which takes place at that moment, of contact between branches of nerve cells, of a nervous current which has been established, etc., etc. Has this communication, this contact, this current, been observed by them through the microscope at the time of its occurrence, or have they even ascertained the existence of an association of ideas? If not, then why do they mention it? Such modes of expressing oneself are mere, meaningless, verbal translations. The actual psychological fact is made to disappear by translating it into another language; nothing is added to our real knowledge, and it would be as well to speak in poetical metaphors as to indulge in this vainest and hollowest of metaphysics.

We may now enter upon the detailed study of psychological methods, most of which are well analysed in the dictionary. Attention may be called to the work of Dr. Fr. Warner under the title of "Psychosis," and to the methods he employs in order to register movements and to interpret them from a psychological point of view. Several authors, particularly Mr. Coupland, insist upon the necessity of studying social life in order to arrive at a correct interpretation of mental facts. "An individualistic psychology, aided by all the resources of the psychical laboratory or clinical experience, would be but a maimed and incomplete psychology; mind cannot be comprehended without a study of its development, and the development of mind implies the action and interaction of minds." Each of these various methods must complete the other.

Consciousness has been studied from various standpoints, for instance, it has been analysed in itself, in its psychological nature, and in its connection with physiological phenomena. It seems to us that in the first essay the author greatly complicates the problem; he speaks of all the phenomena of thought, of comparisons, of ratiocination, of scientific knowledge, which is not a fact of simple consciousness, but is superadded to consciousness properly so called. On the other hand, it appears that the author does not sufficiently take into account the different varieties of consciousness itself. The elementary consciousness, which attends the simplest psychological facts, and the personal consciousness where the idea of the "ego" plays the principal part, should not be absolutely confounded. This distinction is particularly useful for the interpretation of pathological phenomena.

In the second essay the physiological conditions of consciousness are carefully analysed; it is shown that consciousness does not exist without nervous action, but that the reverse is not true and that nervous action may, in appearance at least, sometimes be produced without consciousness. The distinction between the different degrees of consciousness could fairly be applied to this discussion. Authors perhaps too easily adopt the theory of "epiphenomenal consciousness," and do not sufficiently observe that this theory is at variance with their fundamental principle concerning the correspondence which exists between psychological and physiological facts. We will not insist upon the description of the disturbances of consciousness; it is given in the dictionary in a very elementary manner. Let us merely express our amazement at finding that the facts of the double of consciousness, and even the obsessions of lucid madness are being unhesitatingly connected with cerebral duality. For more than ten years these somewhat childish interpretations have been attacked in France and in England, why then are all objections passed over in silence? Let us also express our regret at finding that the numerous researches on subconscious phenomena, and the overwhelming part they play in mental pathology are barely hinted at. Possibly the authors did not deem it expedient to enter deeply into the studies on the pathological alterations of consciousness, and so confined themselves to the description of well-known facts without entering into analyses which seemed to them, perhaps with some reason, still resting upon too hypothetical a basis to occupy a place in a dictionary. The sensations are repeatedly analysed, their laws, their conditions, and their principal modifications are graphically described; the combinations which go to form cœnesthesis, the secondary sensations which produce the well-known phenomenon of "coloured audition," form the subject of interesting articles. The discussions might be more complete, but the leading ideas are sufficiently indicated. But why did the authors shrink from investigating the problem of muscular, or kinesthetic, feeling? The laws of the association of images have been carefully studied, as was to be expected in an English work. Mr. Coupland points out, with great truth, the close analogy which exists between two laws which formerly were kept too much apart; that is, the laws of association by contiguity and by similarity. He recognizes and very clearly demonstrates that association from similarity is in reality nothing but an association from partial contiguity. Some excellent remarks on the association of ideas which formerly had not been united, and on the difficulties of those new combinations should have led him to an investigation of the phenomena of mental synthesis which, as a rule, are so little studied in England. Among the various articles on "Memory," we will call the attention of readers chiefly to a summary of the labours of Mr. Galton on visual memory; and to the processes most frequently used by calculating prodigies. The disorders of memory are well described, and we only regret that the authors did not enter more fully into their psychological interpretation.

It is well known that English psychologists never were addicted to the study of the higher operations of the mind, such as, for instance, judgment, belief, and generalisation. They are always inclined to confound them with simpler phenomena, such as the association of ideas. This tendency appears also in the dictionary, although some exceptions must be made, *e.g.*, for Mr. Coupland's article on "Classification" (38), and especially for Prof. James Sully's remarkable essay on "Attention." Most of the recent experiments concerning attention, its duration and its intermittances, are fully summed up. The theory of M. Sully, which may be summarised thus: "Attention reduces the confused multiplicity of excitations to a relative simplicity," confirms and explains most of the known facts. It was difficult to exhaustively study "Language" in the dictionary; and only the leading ideas concerning its formation, and the disturbances which it presents in the various forms of æphasia, are indicated. But why pass so lightly over the "inner speech" and psycho-motor and Rivesthetic verbal hallucinations, which play a prominent part in mental diseases? As regards the phenomena formerly grouped together under the appellation of "active faculties," we will point out an excellent essay by Prof. Romanes on "Instinct"; some passages by Mr. Coupland on the "Emotions"; and the interesting article by Dr. C. Mercier, on "The behaviour of the insane." In this latter work we clearly perceive how the character of human activity chiefly consists in a continuous effort to adapt oneself to the complex and varied external circumstances; and how, in the mind of the insane, this power of adaptation is subject to metamorphoses and diminutions.

The study of the Will is one of the most difficult topics; too much attention is, perhaps, still bestowed on absolute problems, it would be better to minutely analyse the psychological characters of the phenomenon itself, without paying so much attention to the more or less intelligible theories concerning liberty. This task has been attempted in several articles by Mr. Coupland and Dr. Warner. Perhaps it would have been advisable to lay greater stress upon the considerable part which the idea of personality plays in voluntary actions.

Finally, the disorders of volition are dealt with in a very interesting article by Prof. Ribot; and several essays on suggestions, obsessions and impulses, complete the analysis of voluntary actions. In a rather short article, Mr. Myers tells the history of hypnotism; he lays before us certain, hitherto unknown, details concerning the development of these researches in England. Several authors describe hypnotic phenomena without, in my opinion, sufficiently insisting upon the psychological interpretation of these phenomena, now so well-known and widely discussed.

It is hardly possible to fully review a dictionary, and still more difficult to analyse a whole psychology disseminated through its pages; we do not pretend to pass a final judgment upon the whole work, but merely to express in a few, possibly somewhat superficial, words the general impression which the reading of those articles has produced upon us.

The psychology exposed in this book is, on the whole, that of the English school, as expounded by Prof. Bain and Mr. Spencer. We do not intend here to discuss this psychology; it has advantages as well as drawbacks which are well-known; but we wish to make a general remark about it. It is a general, a theoretical psychology which is applicable to man in his normal state; it does not deal with the diseased individual for the interpretation of whom it is not adapted. Now, the alienist principally requires a pathological psychology, analogous in its main features, to normal psychology, but also laying chief stress upon the points with which he is chiefly concerned. The theories of perception, the transformation of the image into an external object, which is the starting-point of hallucinations, belief, doubt, automatism, &c., deserve a closer study from this point of view. It is not a defect in the dictionary that we are now censuring, but rather a defect in psychology itself. Let us hope that this science will become more and more practical, and more and more capable of rendering to the physician alienist such assistance as he is entitled to expect from it.

5. *Treatment and Care of the Insane.*—Pathology and psychology form only a part of this great work; specialists in lunacy will also find in it much information and many practical hints. Numerous articles refer to the treatment of the insane; among which is a very encouraging essay by Dr. Pliny Earle, on "The Curability of the Insane." Further, the articles upon the psychology of insanity, the treatment of the insane by boarding out, the construction of the asylums themselves, the general and some special methods of treatment of the insane, as for instance, treatment by electricity, which is described with great care. It is, perhaps, to be regretted that the various forms of moral treatment which play so important a part, are so vaguely indicated. It is difficult to gain an accurate knowledge of the special legislation generally very imperfect, under which lunatics are placed. English physicians will be glad to find in the dictionary all the practical information indispensable to them.

Some of the medico-legal discussions have a more general interest, such as those which refer to the responsibility of the insane. The important article by Dr. Orange on "Criminal responsibility in relation to Insanity," sums up all past discussions about this difficult question; it treats of the very controversial problem of partial monomania, and gives the reader a general idea of the principal works upon criminal legislation in its application to the insane.

Dr. Havelock Ellis's article on "Criminal Anthropology" summarises the ideas of Lombroso, of Benedikt, and of Ferri, concerning the relations between crime and insanity.

Such are the principal articles contained in this great work. We have hardly attempted to do more than to point them out to the reader, and give a summary idea of their contents, so as to demonstrate their variety, their importance, and their usefulness to psychologists, physicians and legislators.

The few criticisms in which we have indulged only tend to prove the great interest which we have taken in reading this work.

PIERRE JANET.

Illustrations of the Mid and Hind Brain. BY ALEXANDER BRUCE, M.A., M.D., F.R.C.P.EDIN. Young J. Pentland, London and Edinburgh, 1892.

THIS is an elaborate work on the nerve tracts of the mid and hind brain, and the cranial nerves arising therefrom. It consists of two portions: the first is given over to a description of the author's own observations upon this portion of the brain, the reading of which is rendered easy by a number of valuable schemata: while the second part consists of a series of highly artistic coloured plates, taken from the author's original sections.

In all cases the preparations have been made from the human fœtus; and, except where otherwise stated, from fœtuses at the time of birth. To ensure accuracy in reproduction the plates were taken from the sections, which have been stained entirely by the method of Weigert, under the camera lucida.

Throughout the text there is a digest of the recent literature upon the subject.

The work is divided into two chapters; the first dealing with the cranial nerve nuclei and their connections, the second with the tracts of the medulla, pons and crura.

In the chapter devoted to the consideration of these tracts, prominence is given to certain structures which are still the object of investigation. Reference is made firstly to the upward continuation of the antero-lateral ground bundle of the spinal cord. The anterior ground bundle of the cord is displaced backwards by the inferior olive, so as to become the posterior longitudinal fasciculus of the medulla, and in one of the plates this relation is well shown, the two structures being seen in continuity in a well-planned longitudinal section. The adjacent portions of the lateral ground bundles lie in what Flechsig has designated the inner field of the *formatio reticularis* or area between the roots of the hypoglossal nerve and the raphé; while the outermost of the lateral ground bundles forms chiefly the longitudinal fibre system of the grey reticular formation. The chief of these form the "lateral medullary tract," a strand containing the antero-lateral tract of Gowers, which ends mainly in the lateral nucleus of the medulla. The author considers that the upward continuation of this tract is more likely to be settled by a study of its degeneration than of its development—a view in which we concur.

The author's observations upon the formation of the mesial fillet accord with those of other observers who have worked with this method; its termination, however, being beyond the scope of the work, is still left as a matter of doubt. A relation of some importance between the lateral fillet, and the lateral column of the spinal cord is indicated.

The restiform body is formed of two distinct portions; the external or spinal division being composed of the direct cerebellar tract, of fibres from the post pyramidal and cuneate nuclei of the same and opposite sides, the olivary-restiform fibres, and of fibres from the lateral nucleus of the medulla. The internal or medullary portion is derived from the external nucleus of the vestibular root of the auditory nerve and of a tract from the superior olivary body.

The superior cerebellar peduncle, the inferior olivary body and nucleus dentatus of the cerebellum, the corpus trapezoideum, and the flocculus are each the subject of careful study.

But the chapter upon the connections of the cranial nerves is of greater interest to physicians. Mention is therefore made of the following points which appear to be of clinical importance.

1. *The hypoglossal nucleus.*—The author has traced by longitudinal horizontal sections a fine bundle of fibres running parallel to the raphé from the hypoglossal nucleus towards the knee of the

facial nerve; while a connection between this nucleus and the internal auditory nucleus is also indicated. He confirms the observation originally made by Lockhart Clarke that fibres pass from the hypoglossal nucleus to the issuing vago-accessory nerve roots, and he thinks it probable that this nucleus forms at least part of the motor nucleus of the vagus.

2. *The ascending root of the auditory nerve* (vestibular nerve) is shown to have an important connection with the cuneate nucleus, a relation which does not appear to have been previously pointed out. By this means the vestibular nerve forms important connections with the fillet, the restiform body and the postero-external column of the spinal cord. The author negatives Edinger's view that the ascending auditory root is a "direct sensory cerebellar tract" connecting the roots of the vagus, glosso-pharyngeal and auditory nerves with the cerebellum. It is found to be continuous with and to myelinate at the same time as the anterior root of the auditory nerve.

3. *The facial nerve.*—Without bringing forward any further proof of the several nuclear origins of the fibres composing this nerve, other than that already mentioned, the author merely states the views expressed by Mendel, Gowers and others, and gives a scheme of the probable composition of the emerging root of this nerve, and indicates its tripartite formation from the oculomotor, facial and hypoglossal nerve nuclei.

4. *The sixth nucleus.*—An elaborate diagram is given of the connections of the sixth nerve nucleus, which are stated as follows: (*a*), with the posterior longitudinal bundle of the opposite side; (*b*), with the superior olivary body; (*c*), with the internal and external auditory nuclei and the flocculus of the same side.

5. *The ascending and descending roots of the fifth nerve* are briefly described. The termination of the fibres of the former in the cells of the grey matter internal to the gelatinous substance of Rolando is indicated, but their further relations are still "*sub judice.*" A connection between the substantia gelatinosa and the cuneate nucleus is mentioned, as well as a hitherto undescribed tract of fibres coursing parallel to the ascending root, but on the mesial side of the gelatinous substance. The origin of the fibres of the descending root is stated to be as high as the upper limit of the nucleus of the third nerve.

6. Some space is given to a detailed description of the several groups of cells which form the nucleus of the *third nerve.* The author, who has carefully studied this subject, has very properly adopted a nomenclature in accordance with the position of the

several cell-groups rather than from the name of the observer who originally described them. These cell groups are: (*a*), anterior; (*b*), postero-external; (*c*), median or central; (*d*), postero-median (the pale or Edinger-Westphal nucleus; (*e*), superior (the small-celled nucleus of Darkschewitsch).

We have no hesitation in saying that this work takes high rank among the numerous treatises upon the complicated subject with which it deals. To teachers and students of neurology the book is invaluable; but we also cordially recommend it to physicians who are interested in this subject, by reason of its practical bearing on the study of diseases of the nervous system.

W. A. TURNER.

Abstracts of British and Foreign Journals.

SOME RECENT PAPERS ON NEUROPHYSIOLOGY.

(1) **Einthoven. Broncho-motor Action of Vagus** (*Pflüger's Arch.*, 1892.) An action of nerves upon bronchial muscle has long been an article of medical faith, more especially in the theory of spasmodic asthma, but until quite recently the physiological demonstration of such an action has been very incomplete. Manometric observations of intra-pulmonary pressure have not afforded marked enough results for us to admit bronchial spasm as a really effective phenomenon; the variations of pressure observed by Bert and Franc have been insignificant in comparison with the amount of action that must be supposed to occur in a fit of asthma. The reason of this failure will be evident when we have recognised the principle of the method by which a satisfactory demonstration has recently been given by Professor Einthoven (of Leyden).

This method is as follows: A dog paralysed by curare is submitted to artificial respiration, great care being taken to inflate at regular intervals with uniform quantities of air, a side tube from the tracheal tube leads to a manometer, by way of a special tap which is automatically opened by means of the respiration apparatus for a definite short period of each respiration cycle, thus letting a short puff of pressure into the manometer. If the intra-pulmonary air pressure rises and falls regularly, the succession of puffs produces a level series of manometer effects, but if the bronchial muscle be made to contract by excitation of the peripheral end of the vagus, the puffs occur at greater pressure and the manometer rises. A constriction of minute bronchioles which might cause little or no effect upon a *steady* intra-pulmonary volume or pressure of air, will produce a very evident effect when the rising and falling phases are, so to speak, "tapped" on this "window plan," which is similar in principle to the method of "tapping" an electrical pressure by the rheotome (p. 312).

By these means Einthoven has found: (1) That the excitation of the peripheral end of either vagus causes strong bronchoconstriction, shewn by increase of pressure in, and distension of the lungs; (2) that this action is abolished by nicotin or by atropin; (3) that broncho-constriction is caused by inhalation of CO_2. The broncho-constrictor effect of vagus excitation begins in about 1.2 to 1.3 seconds, and wears off very rapidly during excitation; the effect on the manometer may amount to 175 mm. H_2O or more.

These results, as set forth in Professor Einthoven's paper, fully substantiate this method as a valuable means of studying the action of plain muscle, comparable with the blood-pressure method, and in some respects of superior simplicity.

To many readers the bearing of these results upon the theory of asthma, will be the point of greatest interest. This is dealt with in the two concluding sections of the paper, where the author urges that bronchial spasm is capable of producing acute emphysema, that the respiratory type during an asthma paroxysm is strong, short inspiration, followed by weak long expiration (and not, as usually described, by still more violent expiration), and that *possibly* the subcutaneous injection of atropin may be found of service to cut short a paroxysm. The action of stramonium vapour was not investigated.

Einthoven's paper has given rise to a considerable amount of interest in Vienna. Beer (writing from von Basch's laboratory) published a preliminary communication (*Cbt. f. Physiologie*, 1892, p. 782) as a direct consequence of that paper, and containing several similar conclusions, but laying stress upon the mechanical effects of the circulation. The same author has more recently published a paper of upwards of 100 pages in the 1892 supplement of *du Bois-Reymond's Archiv*.

Von Basch (*Pflüger's Archiv.*, vol. 52, p. 415) and Grossmann (Ibid., p. 567) vehemently criticise Einthoven's criticism of their previous papers. The criticism is very controversial in tone and very obscure in exposition, and does not affect the main point of Einthoven's paper. Grossmann reaffirms von Basch's doctrine that engorgement of the pulmonary capillaries obstructs respiration by distending the alveoli, and quotes figures from Einthoven to show that they prove what his own figures had proved; but on referring to the explanation of them given by Einthoven, and omitted by Grossmann, it will be found that there is no real similarity either in the figures or in the conclusions drawn from them.

(2) **Heese. Influence of the Sympathetic upon the Eye, more particularly upon the Movements of the Iris** (*Pflüger's Arch.*, 1892). The vexed question whether dilatation of the pupil

by excitation of the sympathetic is effected by active contraction of a radially disposed *dilatator pupillæ* or by passive relaxation of the constrictor pupillæ, appears to be finally set at rest by the experiments of Heese. His proof may be thus shortly summed up: graphic records of radial contraction of the iris, caused by excitation of the sympathetic, after removal of the sphincter; ditto of a sector of the iris isolated by two radial incisions. Great care was taken to perfectly immobilise the eye, and the records given are typical of the contraction of smooth muscle—latent period of about 2 seconds, contraction lasting 10 to 15 seconds.

(3) **Langley and Anderson** have given expression to an identical conclusion, based upon similar data. The two papers are quite independent of each other; the date of publication of the German paper is September 16th, 1892; the dates of publication of the English papers are May 14th (communication to the Physiological Society) and October, 1892 (full paper in *Journal of Physiology*).

(4) **Nahmmacher on the Influence of Reflex and Central Excitation of the Optic Nerve upon the Position of the Cones of the Frog's Retina** (*Pflüger's Archiv.*, 1893, p. 375). The experiments on the retino-motor action of fibres in the optic nerve (of which the first results were announced by Engelmann, at the Copenhagen Congress in 1884, *Pflüger's Archiv.*, vol. xxxv.) have, on several occasions, been taken up again in the Utrecht laboratory. Grijns (as reported by Engelmann in Helmholtz's *Festschrift*, two years ago) found on the frog that the retinal current of one eye varies with actinic and chemical stimulation of the other eye. Engelmann, in the absence of any evidence of the possibility of obtaining evidence of reflex electrical variations in different nerves by excitation of other different nerves, concluded that the reflex retinal variation observed by Grijns, depends upon different fibres, *i.e.*, upon the retino-motor fibres inferred to be present in conclusion of his micro-physiological observations on the retinal cones. The observations of Gotch and Horsley to the effect that the excitation of afferent nerves may give rise to centrifugal variations along other afferent nerves, are, however, contradictory of Engelmann's negative assumption; and the experiments of Nahmmachar were undertaken to determine whether the crossed retinal effects are attributable to efferent or to afferent fibres. His conditions of experiment, and his results were briefly as follows:—Rana temporaria; July, August, and September; with and without section of an optic nerve; slight curarisation; total darkness for five

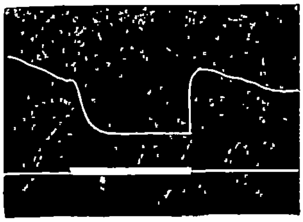
hours; chemical excitation of one retina by a crystal of common salt left in the eye for 10 m. before decapitation; eyes placed in $3\frac{1}{2}$ % nitric acid. Comparison was made between retina of different frogs: (1) normal; (2) injured and not excited; (3) injured and excited; two series of experiments—the first with reflex from eye to eye, the second with direct excitation of the chiasma or optic nerve. As regards the first series, the cones were found to be retracted in thirteen out of fifteen cases of reflex excitation *versus* eight out of fifty-two control cases without excitation (viz., 86.6 *versus* 15.4 per cent.); as regards the second series, the cones were found retracted in forty-two out of fifty-one cases of direct excitation *versus* eighteen out of ninety-two control cases without excitation.

The author's conclusion is that these figures amount to a final proof that the optic nerve contains fibres, the excitation of which causes contraction of the cones of the corresponding retina, *i.e.*, presumably efferent fibres.

(5) **Fritsch,** in the account of his recent observations on electrical fishes (*Du Bois-Reymond's Archiv.*, 1892, Suppl., p. 229), makes remarks on the motor cells of the spinal cord calculated to make us pause before accepting the functional interpretations that have been lately given of the fine anatomy of nerve-cells. The electrical nerves (Mormyrus) arise as unbranched axis-cylinder processes from large multi-polar cells (100 to 500 μ) that occupy the entire transverse section of the grey matter. These unbranched processes acquire a medullary sheath while still in the cord, and are thus readily distinguished from the "protoplasmic" processes, which unite the cells into a closed plexus "as if for a common function." He proceeds to say: "This discovery gives positive proof that protoplasmic processes serve to unite nervous elements, and disproves the supposition of Golgi and of his followers, to the effect that the axis-cylinder-process alone is of 'nervous character.'"

(6) **Kaiser. Interference-phenomenon in the Nerve-Muscle Preparation** (*Zeitschrift f. Biologie*, 1892, *p.* 417, *vol.* 28). An extremely interesting experiment, exhibiting what at first sight it would appear most natural to designate as an inhibitory phenomenon, has recently been published by Kaiser. The experiment is easily repeated and appears to be a suitable one to include among practical exercises such as are drawn up for advanced students. It may therefore suitably be described in the form of an instruction to such students about to repeat it.

A nerve-muscle preparation is made, and enclosed in a moist chamber; the muscle is connected with a recording lever and weighted by 20 grms.; the central end of the nerve is laid across ordinary platinum electrodes from an induction apparatus; a portion of the nerve nearer to the muscle is made to hang in a loop and brought into contact with strong glycerine. As the effect of this glycerine stimulus, the muscle after a time becomes tetanised; when this tetanus has fully developed an interrupted current of moderate strength is thrown in. During its passage the tetanus is cut down, as shown in the accompanying figure taken from among the illustrations given by the author.



(7) Piotrowski (*Centralblatt f. Physiologie*, 1892, p. 604, *Journal of Physiology*, 1893, p. 163), working in the Cambridge Laboratory, repeats and extends Biedermann's observations on the muscles of the crayfish claw. With weak currents the abductor muscle give contraction, with strong currents relaxation. The adductor muscle does the reverse; *i.e.*, relaxes with weak and contracts with strong currents. These antitonic effects, best brought out when the disposition to tonic contraction is most pronounced, *i.e.*, in the summer, they are best demonstrated on the abductor; and in all such experiments the antagonist muscle is of course divided. By weak single shocks he obtains double effects of a similar character, viz., a preliminary relaxation preceding the contraction.

He regards these effects as being in harmony with Hering's theory of dissimilation and assimilation, *i.e.*, that the relaxant effect of stimulation is a true inhibitory effect. In conclusion, he remarks: "The process of inhibition has its seat in the muscle itself. Biedermann's histological researches justify us up to a certain point in assuming that the stimuli of the two processes [D. and A.] are conducted through two kinds of nerve-fibres, and appear to confirm Gaskell's view that every tissue possesses katabolic and anabolic nerves, of different structure and of different origin."

(8) **Boruttau** re-examines the point whether there is a physiological "lost time" in the motor end-plates (*Du Bois-Reymond's Archiv.*, 1892, p. 444,) and confirms the conclusion of Bernstein that there is. He ascribes the contradictory result of Hoisholt to the fact that the latter compared the effects of direct and indirect excitation of muscle with stimuli of insufficient strength. Piotrowski, experimenting on the adductor muscle of the crayfish claw, comes to a similar conclusion as to the existence of a physiological lost time.

(9) **Sherrington** (*Proc. R.S.*, Feb. 2 and April 15, 1893). With reference to the correlated action of antagonist muscles Sherrington's latest observations on monkeys are of considerable neurological interest.

He finds that the physiological state of the extensor muscles of the thigh (as tested by the knee-jerk) is influenced by the physiological state of the flexor muscles; thus paralysis of the flexors by nerve-section entails exaggerated response of the extensors, while that response is diminished or abolished during artificial contraction of the flexors, or by their passive extension, or by central excitation of their nerves; and he finds that by the proceeding last named the reflex tonus of the extensors is increased.

Still operating on the monkey he gives another and somewhat different illustration of the mechanism of functional correlation. After section of the 3rd and 4th cranial nerves of the left side, leaving therefore the 6th nerve and external rectus muscle alone active upon the left eyeball, he finds that appropriate cortical excitation will still produce conjugate deviation of the eyes to the right, viz., that presumably the left external rectus muscle was inhibited by the cortical excitation. This, it need hardly be said, is not offered as proof that the 6th nerve has an inhibitory or anti-motor action on the external rectus muscle; it is a physiological illustration of arrest of action by cerebral excitation, at a particular part of the muscular periphery experimentally picked out from the group of physiological movements in which it normally plays part. The experiment shows that in the lateral deviations of the eyes, the opposing muscles do not contract but give way, and it is left an open question whether the interference phenomenon is cortical or bulbar or muscular.

(10) **Charpentier** (*Archives de Physiologie*, 1892, p. 541), in prosecution of his optical investigations, publishes two very

beautiful and easily repeated experiments demonstrative of a slow oscillatory physiological process in the retino-cerebral apparatus.

(1) A disc ¾ black and ¼ white, revolving once in one to two seconds, viewed by direct sunlight, exhibits on the white quadrant a narrow black sector near the black edge that has just passed in front of the eye, and separated from that edge by a narrow white sector. This is presumably due to a rebound effect of the nature of an excitatory oscillation in the retino-cerebral apparatus; the first effect at the arrival of the white border is white, but this first effect is followed by an after-effect that is black, even *during* the continued white stimulus. Estimating from the rate of revolution and the apparent position and extent of the black band in the white quadrant, the positive and negative phases of the excitatory oscillation are found each to have a duration of 0.015 sec., indicating an oscillation frequency of 33 per cent. It is probably in consequence of an oscillation of this nature that a single bright stimulus causes a double or reduplicated bright sensation.

(2) A black disc, with narrow open sectors, revolving in front of the eyes, fixed upon a white surface illuminated by direct sunlight, gives rise to the sensation of a magnificent purple-violet field, when the rate of revolution is such that the retina is stimulated between 40 and 60 times per second, *i.e.*, when each stimulus occurs during the negative after effect of the preceding stimulus. At higher and at lower frequencies the field is colourless, and at the optimum frequency a white or yellowish space is seen in the centre of the purple field, corresponding with the yellow spot, in which the retinal purple is deficient. Charpentier is of opinion that the coloured sensation is due to entoptic vision of the retinal purple.

By calculation from another series of experiments, Charpentier finds that the excitatory oscillation, with a period of 0.028 sec. and a frequency of 36 per sec., is propagated from its seat of origin with a retinal velocity of 72 mm. per sec., and a wavelength of two mm.

(11) **R. Dubois,** *Anatomie et Physiologie de la Pholade Dactyle*, Paris, 1892.—This important monograph is full of observations on the dermatoptic properties of the integument of a well-known mollusk (Pholas Dactylus) that are of great importance to the general neurologist; the author develops at some length a new theory of retinal excitation, based upon his own observations, and reminding one of the hitherto isolated facts first pointed out

by Engelmann and van Genderen Stort with regard to the retractility of the retinal cones of the frog's retina. We cannot in a brief notice do justice to this memoir, but we may give the following summary of one of the author's main conclusions. There are very remarkable structural and functional analogies between the retina and the integument; an external layer of pigment-cells is connected with a superficial layer of muscular fibres that contract in response to luminous stimuli. This initial local contraction constitutes a mechanical stimulus to subjacent nerve-cells, and is followed by a secondary total contraction of the longitudinal musculature of the siphon. Histologically the dermic element excitable by light is composed of: (1) pigment-cell, (2) contractile fibre, and (3) nerve-cell. Functionally the peripheral stimulus is a dermic phosphene analogous with a tactile phenomenon, viz., a mechanical excitation of the nerve-cell, by a movement of the contractile fibre, in consequence of a photo-chemical change in the pigment-cell.

A.D.W.

A Study of the Artefacts of the Nervous Sysem. By IRA VAN GIESON, M.D. (Reprinted from *New York Medical Journal*, September and October, 1892.)—Under this title Dr. Van Gieson gives a study of the "Topographical alteration of the grey and white matter of the spinal cord caused by autopsy bruises."

The word "artefact" has been ingeniously coined to avoid the use of the originally similar word, "artifice," which, owing to the common tendency of words to degenerate or take a less worthy meaning as language develops, would imply some disingenuousness on the part of the observer.

The author believes that "true heterotopia" of the grey matter may exist, but that it is extremely rare. He admits the existence of only six recorded cases, of which he gives a short *resumé*. In all these the displaced grey matter was apparently from the posterior horn and in the posterior columns. They were very minute and are presumed to be examples of true heterotopia because of the absence of any bruising of the surrounding white matter. We fail, however, to see in what respect these small displacements differ from the larger artificially produced alterations, except in degree. The grey matter must be supposed to be more diffluent than has hitherto been suspected, or the remarkable appearances produced artificially by Dr. Van Gieson would be impossible. We now come to the very interesting part of the work devoted to the description of the deformities produced by bruising. A large number of drawings are given of sections from cords, first accidentally injured in removing, and secondly subjected after removal to every form of experimental injury. The effects produced are very remarkable, and the most prejudiced reader cannot fail to be struck by the close resemblance of the appearances to those in the recorded cases of so-called heterotopia. Even such effects as duplication of the cord have been produced artificially and figured by Dr. Van Gieson.

Then follows an analysis of the recorded cases of heterotopia, with reproductions of the plates, which Dr. Van Gieson condemns as artificial and not pathological at all.

The impression left on the mind of the reader is that there is no such thing as heterotopia, and the practical lesson to be learnt is, in removing a spinal cord never touch, bend, or twist it, and lift it only by its dura mater.

H. H. TOOTH.

Die Circulation im Gehirn und ihre Störungen. By Dr. RICHARD GEIGEL. *Virchow's Archiv*, vol. cxix., p. 93.

Die Regulirung der Blutbewegung im Gehirn. By Dr. B. LEWY. *Virchow's Archiv*, vol. cxxii., p. 146.

"Experimentelle Beiträge zur Lehre von der Blut-Circulation in der Schädel und Ruckgratshöhle." By Professor HUBERT GRASHEY. *J. F. Lehmann, München*, 1892.

Geigel begins by pointing out that Althann, of Dorpat, writing in 1871, demonstrated that the condition of fulness of the brain vessels was no measure of the good or bad blood supply of the nervous elements; but that the point of importance is the ease with which oxygen is brought and carbonic acid removed from these elements.

This depends on two things :—

1. The chemical constitution of the blood.
2. The quantity of blood that passes through the capillaries in a given time.

Geigel proposes to designate a satisfactory flow of arterial blood through the capillaries as eudiæmorrhysis, too little as adiæmorrhysis—true anæmia cerebri, and too much as hyperdiæmorrhysis—true hyperæmia cerebri.

He then states a proposition as follows :—

Let g = the velocity of the circulation of the blood in the capillaries of the brain. This is directly proportional to the arterial pressure a, and inversely proportional to the resistance w. Therefore $g = \frac{a}{w}$

But w depends directly on the amount of the intra-cranial pressure = d, therefore $w = f(d)$ and $g = \frac{a}{f(d)}$

And Fick has shown that the intra-cranial pressure is equal to the intra-arterial pressure, less the resistance which the tension of the arterial walls oppose to it, therefore $d = a - s$ and $g = \frac{a}{(f\,a-s)}$

From this it follows: 1st, that if the contraction force of the arterial wall gets less; intra-cranial pressure = d = a - s will get greater and g will get less, that is, the circulation will alter in the direction of adiæmorrhysis: in other words, dilatation of an artery causes anæmia, not hyperæmia.

2nd, if the tension of the arterial wall becomes greater d = a - s will become less and g will get greater, that is, the circulation will alter in the direction of hyperdiæmorrhysis, so that spastic contraction of the arteries of the brain will increase the velocity of the bloodflow through its capillaries.

Again, suppose the heart acts more powerfully, raising intra-arterial pressure, while at the same time the arterial walls increase their contraction, intra-cranial pressure will be the same, or greater or less, according as the arterial contraction equals, or is less or greater than the rise of intra-arterial pressure.

Hence, with increased contraction of arterial walls intra-cranial pressure decreases and capillaries widen. On the other hand, with diminished contraction of arterial walls intra-cranial pressure is raised, the capillaries are compressed, and the amount of blood circulating through them is diminished.

The writer points out that the symptoms of cerebral pressure are really due to the interference with the circulation which the pressure produces, and are not the direct result of the pressure on the cerebral substance: for the brain substance will bear the pressure of two atmospheres and more without harm, provided its circulation is not thereby interfered with.

The supply of oxygen and removal of carbonic acid are the points of importance, and these are equally interfered with by high intra-arterial pressure and by anæmia; hence the symptoms of these two conditions may much resemble each other.

He applies the same reasoning to the effects of embolism or rupture of a vessel.

By an ingenious arrangement of tubes, partly glass and partly elastic and expansile, passing through a closed container full of water, he shows that when one tube is blocked to represent an embolus the flow is contemporaneously diminished through the other tubes which are not blocked, owing to the general fall of pressure in the closed container produced by the contraction of the vessel that is blocked.

Thus embolism of one intra-cranial artery produces a temporary diminution of circulation in all the other intra-cranial vessels, and thus "adiæmorrhysis" is the cause of the apoplectic shock of embolism.

The rupture of a vessel produces a similar general adiæmorrhysis by raising the intra-cranial pressure, for in normal conditions $d = a - s$, but when a vessel is ruptured s is more or less completely removed, and $d = a$, and the resulting adiæmorrhysis is the cause of the apoplectic shock, and this will be more severe the more rapidly d is made = to a.

He argues also that the same laws apply to the cranium of a child with the fontanelles open; that spastic contraction of arteries causes hyper-diæmorrhysis and paralytic dilatation adiæmorrhysis because the dura mater keeps up the internal pressure and acts very much as the closed skull in the adult.

Lewy takes a somewhat different view of the matter, for though he acknowledges that an intra-cranial artery cannot expand without taking space from other vessels, he points out that the space thus taken is so small that in physiological conditions it affects the cerebro-spinal fluid, but not the capillary circulation at all.

Again: when an artery enlarges, the blood meets with less resistance in passing through it; and it is possible that the lessened resistance in the artery more than compensates for any slight increase of resistance in the capillaries due to the expansion of the artery, and he points out that Geigel has overlooked this.

Conversely, contraction of an artery may increase the resistance more than the corresponding relaxation of capillaries diminishes it.

When the arteries all enlarge together, the lymph gets out of way and the capillaries are so numerous that they will bear a large amount of compression before they are so far closed as to hinder the circulation; and under these conditions narrowing an artery diminishes the blood stream and widening an artery increases it, so that arterial hyperæmia is possible. But beyond a certain point this does not hold, for if we imagine the arteries to enlarge so much that the veins are pressed flat absolute stasis will result: if the arteries now begin to contract, passage of blood will begin again and increase, and thus a narrowing of the arteries produces hyperæmia.

If, however, part of the intra-cranial space is taken up by a tumour, or again, if a large number of capillaries have been destroyed by injury or inflammation, then a smaller amount of arterial enlargement may seriously interfere with the capillary circulation.

Lewy thus acknowledges that Geigel's argument may hold for certain pathological conditions, but denies that it does so for physiological conditions.

He further points out that when the skull is open the brain is seen to undergo an increase of volume corresponding to each systole; but when the skull is closed there is no increase of volume, but the pulsatory quickening of the blood stream is carried on and becomes visible in the veins.

Lewy, as we have seen, considers that the arteries may possibly enlarge so much as to compress the veins and produce stasis; but Grashey shows that this result may ensue, not so much from dilatation of the arteries, as from rise of intra-arterial pressure.

When this pressure rises beyond a certain height the central veins begin to vibrate, and then the amount of blood streaming through is decidedly reduced. At this point it is possible that the symptoms of pathological brain pressure begin.

At this point dilatation of an artery will still further increase the pressure on the veins and do harm, so that here, to some extent, Grashey agrees with Geigel.

In other points he disagrees with Geigel, for he points out that hyper-diæmorrhysis is not proportional to arterial contraction, for if the spastic contraction of arteries is very great, the blood stream may obviously be stopped altogether: again, contraction or dilatation of arteries must obviously influence intra-arterial pressure, but Geigel argues as if this always remained the same.

Contraction of an artery diminishes the pressure in it, dilatation of one increases the pressure in it; for this reason dilatation of an intra-cranial artery increases the pressure on the veins and does harm, while the local application of cold may do good by contracting the vessels.

According to Grashey, the quantity of blood that streams through the cerebrum in a given time rises with the pressure in the aorta up to a certain point: but when the pressure rises still further the central veins (at or near their point of entry into the sinuses) begin to vibrate, and the amount of blood passing through them is greatly diminished. The vibration appears to be due to the alternate contraction or dilatation of these veins according as the pressure inside them overcomes, or fails to overcome, the compressing force outside them.

Under these conditions the veins behind the point of compression, and the capillaries behind them, are overfilled with blood, which however is able to move but slowly, and the conditions are thus unfavourable for the nutrition of the brain.

Grashey further believes that the stasis thus brought about may cause an increase in the quantity of the cerebro-spinal fluid, and this will still further add to intra-cranial pressure, so that things tend to get worse and worse once this vicious circle is established by the pressure of high arterial tension; and mere removal of high arterial pressure may not now suffice to remove all the symptoms of intra-cranial pressure.

What is said above of the effects of dilatation of a cerebral artery is obviously true of a local dilatation only; for when the arteries all over the body dilate a fall of blood pressure results and the pressure on the intra-cranial veins is diminished.

All these writers agree that the quantity of blood in the brain is of little consequence compared with the freedom of its circulation, and the ease with which it can give out oxygen and take up carbonic acid. And two of them agree in believing that high intra-cranial pressure may compress the veins, and produce stasis and hyperæmia in the smaller veins and capillaries behind them, and that such a condition is unfavourable for the nutrition of the brain even though its vessels now contain an unusually large amount of blood.

A. HAIG.

Contribution a l'étude Anatomo-pathologique et Clinique des différentes Variétés de Cécité Verbale, par M. J. DEJERINE (*Mémoires de la Soc. de Biologie, Séance du 27 Février*, 1892).

This is a most interesting and instructive case of word-blindness. An elderly man, intelligent and well educated, suddenly became unable to read. This brought him under the notice of M. Landolt, who later, sent him to M. Dejerine. He displayed no noteworthy error of refraction, and his direct vision was fairly acute, but he had "*une hémiopie*[1] *homonyme latérale droite,*

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[1] The left halves of both retinæ were affected. It seems desirable here to allude to the extremely unsatisfactory state of our nomenclature of symptoms of this class. With some writers hemianopia and hemiopia are to be interpreted with reference to the retinæ. Thus, for example, *left* hemianopia would signify paralysis of the left halves of both retinæ. With others the same terms are to be interpreted with reference to the field of vision, and *left* hemianopia would then imply darkness of the *left* half of the visual field, and so would connote paralysis of the *right* halves of the retinæ. Those who employ hemianopia in the former of the two senses not infrequently use hemianopsia, to be interpreted as referring to the field of vision. But even were this distinction between left hemianopia and left hemianopsia uniformly observed, there yet remains an objection to the use of the words, namely, that there is no etymological distinction between them to warrant their employment to connote opposite conditions of the retinæ. Analogy with myopia and kindred words may appear a justification for hemianopia, denoting half-retinal blindness; but the ambiguity arising, both from its variable significance, and from its likeness—in sound, as well as in appearance—to hemianopsia, is a real, and not merely an æsthetic, difficulty attending its use. Now amaurosis is already in vogue as a term restricted to a retinal condition,

qui n'est pas absolue," and which was accompanied by a co-extensive, and absolute achromatopsia. Further examination revealed alexia, not merely of words, but of letters and musical signs as well; with the last of which he had previously been unusually familiar. Yet he could recognise and name without delay or error all other familiar objects, drawings not excepted. He could write, both spontaneously and from dictation, almost as well as before his trouble, but was totally unable to read what he had written. Copying, since he could not read, was next to impossible; but he succeeded by literally *sketching* each letter to transcribe slowly, and somewhat illegibly. Cyphers, however, he could read, write, and copy with comparative ease; and he was able to perform easy arithmetical operations with accuracy, though with extreme slowness. His hearing was acute, and in every respect normal; his speech in no way defective; and his powers of *mimique* unimpaired. He had neither hemiplegia, nor ataxy, nor were there any sensory symptoms beyond the visual troubles above described. These symptoms persisted without marked alteration for four years, during which he was under constant observation. Ten days before death, he was suddenly seized with total agraphia, and paraphasia, without any word-deafness, and without any loss of his faculty of *mimique*. There also appeared, transitorily, motor and sensory troubles in the right limbs, and also a complete *Seelenblindheit*, which, too, disappeared before death. The patient sank finally into coma, which passed gradually into death.

At the autopsy, two lesions were discovered in the left hemisphere; the one, of long standing, involved the extreme hind end of the hemisphere; the other, a recent one, being situated more anteriorly. The former lesion occupied, externally, the posterior extremities of the first and second occipital convolutions and the sulcus between them; intero-inferiorly, it involved the base of the cuneate lobule, the walls and floor of the calcarine fissure, and the posterior end of the occipito-temporal gyri, leaving however the limbic lobe intact. The optic radiations were much degenerated, and atrophied, as was also a portion of the splenium of the corpus callosum. The recent lesion implicated the supramarginal, and angular gyri, and the second annectant gyrus. It extended inwards as far as the ependyma ventriculorum. The rest of the brain was normal.

The account is well illustrated by wood-cuts, and to it the reader is referred for fuller details. The association of the old lesion with hemi-amblyopia and cæcitas verbalis *sine* agraphiâ, is most interesting, and it is believed that the author's case is the

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and hemi-amaurosis, besides being unambiguous, is not objectionable from a classical standpoint. Scotosis, too, from its sisterhood to scotoma, a term confined to a condition of the visual field, is familiar to all; and hemi-scotosis is as safe from misapprehension as is hemiamaurosis. It would be better, therefore, both from the utilitarian and from the æsthetic point of view, to designate such a symptom *left* hemi-amaurosis, or *right* hemi-scotosis, expressions both of which connote blindness of the *left* sides of the retinæ. There is no objection, however, to the employment of the convenient adjective *hemiopic* provided that the paralysis be accurately defined by less ambiguous terms.

first such, in which an autopsy has been obtained, and in which therefore the lesion has been described. It cannot, however, be regarded as a strictly unilateral lesion, for though the right hemisphere is described as "*intact à la surface, et sur les coupes,*" a degeneration of the splenium is noted, which presumably extended into the right optic radiations, and therefore constituted a sub-cortical lesion in the right hemisphere. The connection between the recent lèsion and cæcitas verbalis *cum* agraphiâ is accentuated by references to like lesions, associated with similar symptoms, recorded by M. Dejerine himself, Berkhan, and Serieux. But the *necessary* association of "*angular*" lesions with these symptoms is most difficult of proof. For example, in the case in point, the softening reached inwards as far as the ventricle, and so was, in effect, not merely a parietal lesion, but a sub-cortical occipital lesion as well, involving as it must have done many of the optic fibres in their path from the internal capsule to the occipital cortex. The history, too, of brain-physiology, reminds us of the tendency to ascribe to the gyrus angularis, an importance possessed to a greater extent by the regions posterior to it. And consideration of the anatomical relations of this portion of the encephalon, further discourages belief in the exclusive part played by the angular lesion in such cases. For, any lesion that destroys, to its full depth, the convolution in question, lies alarmingly near the fibres of the optic radiations, which at this level have not diverged to any great extent, and which consequently would be injured in large numbers, by a very slight extension of the lesion inwards. Moreover, and this especially in the central nervous system, the apparent, molar, anatomical confines of a given lesion, are by no means its true, molecular, physiological limits. Bearing in mind then, the difficulty of assuring ourselves that a lesion apparently limited to the angular convolution is really confined to that gyrus, and keeping in view the direction taken by erroneous notions in the past, it seems more reasonable to recognise that simple alexia and hemi-amblyopia, and the same symptoms plus agraphia and paraphasia, may result respectively from less extensive and more extensive posterior lesions, than to assume that they correspond to "*deux lésions anatomiquement distinctes.*" It would be interesting to learn whether the final symptoms included any aggravation of the hemi-amblyopia.

In conclusion, we would express our real appreciation of M. Dejerine's valuable contribution to brain-literature.

William Legge Symes.

We are glad to announce that the *Report of the Proceedings of the Congress of Experimental Psychology* held in London in August last, is being published by Messrs. Williams & Norgate, of Henrietta St., Covent Garden. The price to others than members will be five shillings.

BRAIN.

PART III., 1893.

Original Articles.

SOME POINTS BEARING ON THE ASSOCIATION OF SENSORY DISORDERS AND VISCERAL DISEASE.

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In the following pages I deal in a somewhat discursive and desultory manner with the result of several years' enquiry into the symptoms evoked in the sensory nervous system by disease of the viscera. For many years the subject has been present in my mind, and although for a long time I could not appreciate the value of all the phenomena, yet by carefully noticing each circumstance in every case, and by grouping the symptoms together, I formulated for myself certain tentative laws of a somewhat vague nature, trusting that with extended experience these would become more defined and be of a less general nature. What I have published on this subject[1] does not fully accord with the results obtained by Dr. Head,[2] and although in many respects his results are more reliable than mine, there are a few points on which we are in conflict. These points may seem to be here unduly emphasised, but my object in stating them is that a subject of such importance and of such practical utility should be established on a basis which cannot be gainsayed, and that the views of Dr. Head will be enhanced if he is able to rebut objections. On the other hand, these views may be all the better for being modified if they are found irreconcilable with fact. While I, therefore, take exception to some of his conclusions as being too exact, yet I heartily congratulate him on a brilliant and valuable piece of work, and only regret that space does not permit me to emphasise the many points in which my evidence fully supports his conclusions.

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[1] *Medical Chronicle*, August, 1892.

[2] Brain, Part I. 1893.

In pursuing the study of this subject it is necessary to divest the mind of all preconceived ideas bearing upon it and to note carefully every individual circumstance, however contrary it may appear to one's expectations and desires. One great difficulty is the unreliability of the patient. Pain of the most excruciating kind leaves in the mind but a very imperfect recollection of its exact site. In all cases the description of the pain prior to examination should be received with great caution, and instructions given to note accurately the exact site during an exacerbation or recurrence. In the latter case trustworthy accounts can generally be received even when the observer is not present.

I must confess I have also found it necessary to put a control on my own observations, by having them verified by a colleague. In making my observations I invariably had the most striking and important cases examined independently. Such phenomena as my colleagues could not verify I rejected, for if these sensory symptoms are to be of value they must be capable of being elicited by observers free from bias. And further, cases should not be recorded based upon recollections alone, for it is wonderful how far from the truth the memory may wander, when there is a favourite theory to be advanced.

I.—The Pain in Visceral Disease is a referred Pain.

When I had for some time, in systematically examining patients, employed the site of pain as an important aid to diagnosis, I gradually became impressed with the apparent fact that there was little or no evidence pointing to the situation of the pain in disease of the viscera, being situated in the organ affected. This view was strengthened by the results of *post-mortem* examinations in two cases of gastric ulcer, where the sites of the ulcers bore no relation whatever to the seat of pain during life. When Ross[1] published his highly suggestive paper, I was greatly helped in obtaining an explanation of many of my observations. In conversation with Dr. Ross I dissented from his view of the so-called splanchnic pain, maintaining that there was no evidence whatever in support of it. He stoutly maintained the contrary opinion, and used as an illustration a case of gastric ulcer that he had then under observation, in whom there was a pain of great severity localised in a limited area. He said he felt convinced that, were he to push a long needle into this painful spot, the needle would inevitably pierce the ulcer. In opposition to this view (which is very widely held), I urged the fact that whilst the site of pain was absolutely stationary, the stomach (and its ulcer) executed movements of a considerable excursion, both by reason of the musculature of the stomach itself, as well as by the mechanical movements of respiration.

My attention being somewhat accidentally called to the presence of hyperæsthesia in a patient recovering from an attack of gall-stone colic, I made careful search for this phenomenon in other cases, and collected a large number of illustrative cases, intending in the first instance to submit them to Dr. Ross as evidence in favour of the somatic origin of his splanchnic pain. But this was never done, on account of his painful illness and deeply regretted death.

The sensory phenomena, besides pain, when present, exhibit an increased irritability of the peripheral nerves. This is evidenced more particularly by cutaneous hyperæsthesia, and exalted sensibility of certain sub-cutaneous tissues of the external body wall. The first of these can be demonstrated by the usual methods for testing the hyperæsthesia of the skin, while the latter can be elicited by lightly grasping the various structures through the skin between the thumb and fore finger. There can occasionally be demonstrated a difference in the sensibility to this stimulation in the various sub-cutaneous tissues. Thus, when no hyperæsthesia of the skin exists, as tested by lightly stroking with a blunt pointed instrument, when the skin is grasped in the above manner, abnormal tenderness may be elicited. Similarly when the skin is not unnaturally sensitive the sub-cutaneous fat or mammary glands may be so. And again, while these structures may exhibit no abnormality, certain muscles may be extremely hypersensitive. Further, distinct tenderness may be found in pressure over certain vertebræ, and that when there is no increased sensitiveness of the tissues interposed between the vertebræ and pressing thumb. These tender vertebræ frequently afford very valuable evidence of the organ diseased. When hyperæsthesia of the skin is present along with this tenderness of the sub-cutaneous tissues, the two areas are not co-extensive—the tenderness of the sub-cutaneous tissues generally being spread over a larger area than, and even manifested at some distance from, the cutaneous hyperæsthesia. In such cases, if the nerve supply of the organ affected be carefully enquired into, an explanation will be generally found in the fact that the nerves supplying the organ are in connexion with the spinal nerves at different levels of the cord.

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[1] Ross, J., "On the Segmental Distribution of Sensory Disorders," BRAIN, January, 1888.

Pain, when felt by the patient in whom there is also increased sensitiveness of certain areas of the skin or sub-cutaneous tissues (limiting the term sub-cutaneous tissues to the structures of the external body wall), is generally situated within the area of hypersensitiveness. Now as these areas are these to which Ross usually referred his splanchnic pain (the pain in the organ itself), it seems but reasonable to expect that the pain would be produced in the nerves thus abnormally excited. That is to say, if the roots of certain nerves are excited to a degree that produces this increased sensitiveness of the external body wall, it may be reasonably assumed that the irritation of the nerve roots also produces the pain which is felt in the area of their peripheral distribution. That a pain can thus be referred has been accepted by many writers, and Ross adopts it in the explanation of the origin of his somatic pain. The only difference between this referred pain—the somatic—and the so-called pain of the organ itself—the splanchnic—is one of locality, the latter having its seat in close relation to the situation of the organ affected. If we take the pain of renal calculus as an illustration, it will be found frequently that the pain extends from the lumbar region round the side and down the abdominal wall slanting inward to the scrotum with, it may be, retraction of the testicle. As it is known that the ureter occupies a somewhat similar situation, it is frequently assumed that the pain in such a case is felt in the ureter itself. But the 12th dorsal nerve in its peripheral distribution follows a somewhat similar course, and frequently after an attack of renal colic, the skin here is hyperæsthetic. It seems, therefore, much more reasonable to assume that the pain here is in reality due to some irritation at the root of the 12th dorsal nerve, and that the pain is felt in the area of cutaneous distribution in accordance with the law of eccentric projection of pain. This assumption is all the more reasonable when there is evidence of the irritation having extended to more distant nerve roots, as when the pain and hyperæsthesia extend to the regions of distribution of the 1st and 2nd lumbar nerves, and even having affected the motor centres, as evidenced by the retraction of the testicle, and feeling of loss of muscular power in the muscles supplied by those nerves.

But it is in the study of the pain in heart affections that the most striking evidence is furnished in favour of the view that the pain in visceral disease is not located in the organ. Many precise observations have been made supposed to demonstrate the sensitiveness of the organ. Walshe[1] and Balfour[2] speak of the heart becoming painful, and Peter[3] has assumed that he could demonstrate this sensitiveness by exerting pressure on the organ. But in such cases where the exertion of this pressure elicits pain it can be readily demonstrated that the pain is really elicited from stimulation of the external tissues.

The hyperæsthesia of the skin associated with attacks of angina pectoris is no new thing, several writers mentioning the observation. It is needless to affirm, however, that a great many cases of angina pectoris suffer typical attacks without any symptom of peripheral hyperæsthesia, either of the skin or subjacent tissues. Such a case presented itself to me a short time ago where the patient died in the second attack of pain. In him no other sensory abnormality whatever could be elicited. The pain was situated across the lower third of the chest, extending slightly to the left. In such a case one might assume that the pain was undoubtedly in the heart. But is it so in reality? During the period that a patient suffers from an attack of angina pectoris the heart's movements still continue. The movement of such an abnormally sensitive organ would surely cause some variations either in the intensity or in the situation of the pain, and of this there is no evidence. Further, the pain of angina pectoris very often extends over a greater area than that covering the heart. In many cases this extension of the area takes place in a manner very instructive. In the first instance the attacks of pain may be limited to the præcordial region. As they increase in severity they extend further round the left chest and towards the arm-pit, and it may be down the inside of the left arm. The pain appears to be exactly of the same nature in the more distal parts as it is when it is located in the præcordia. It is a mere assumption to say that in the one instance the pain is in the heart, and when felt in more distant regions that it is due to reflex irritation of the spinal roots, particularly when the cutaneous area over which the pain is felt is supplied by nerves whose spinal roots are in immediate contiguity. This is more strikingly demonstrated when hyperæsthesia of the skin is likewise present. In such a case the pain is situated within the hyperæsthetic area, and this area can be demonstrated to be the region of cutaneous distribution of the 4th, 3rd, and 2nd dorsal nerves. The pain may even not be felt in the region of the heart at all. From the study of a series of cases extending over a period of years during which I have observed the attacks of angina pectoris first appear and develop, I have been able to observe a great diversity in the modes of attack. Although the symptoms of pain are usually præcordial at first they are not invariably so. In one patient whom I have had under observation for two years with symptoms of aortic stenosis, the pain and hyperæsthesia in the first instance were situated over the anterior axillary wall and in the arm-pit. Lately the attacks have developed with extreme severity, and the hyperæsthesia now embraces the upper part of the left chest with the inner half of the left upper arm, the ulnar half of the forearm, and the little finger. There is also extreme sensitiveness of the left sterno-mastoid and trapezius muscles. This distribution of the pain and hyperæsthesia is so characteristic, as to leave no doubt of the distinct connection of the cardiac nerve supply with the nerve roots in the spinal cord supplying those hypersensitive structures. Thus the hyperæsthesia of the chest and arm is practically the field of cutaneous distribution of the upper four dorsal nerves of the left side, the roots of these nerves being connected with the sympathetic fibres that supply the heart. The tenderness of the sterno-mastoid and trapezius muscles, indicate irritation at the root of the spinal accessory nerve, and it apparently receives the stimulation from the heart through the medium of the vagus, the roots of the vagus and spinal accessory being connected. The sensory phenomena exhibit great variation in the locality of this distribution. Sometimes the dorsal nerves show no cutaneous hyperæsthesia, while the cervical nerves do. I have at present under observation a patient who has marked attacks of angina pectoris, and these I have noted developing during a number of years. In her there is very slight tenderness of the deeper tissues of the thoracic wall, whilst the left sterno-mastoid and trapezius muscles are extremely tender and the hyperæsthesia of the skin is limited to the left side of the neck, in an area corresponding to what I take to be the peripheral distribution of the 3rd cervical nerve.

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[1] Walshe, W. H., "A Practical Treatise on the Diseases of the Heart and Great Vessels," London, 1873, 4th edition, p. 208.

[2] Balfour, G. W., "Clinical Lectures on Disease of the Heart and Aorta," London, 1876, p. 274.

[3] Peter, M., "Maladies du Cœur," Paris, 1883, p. 36.

But while most of the cases show the pain limited to, or originating in the præcordial area, other rare cases, manifest attacks of angina pectoris, which have no relation to the chest wall. In such cases the term angina *pectoris* is certainly a misnomer, but taking the term to imply that severe pain associated with disease of the heart, it will be found to appear typically in regions distant from the heart. The following case is such a striking illustration of this, that on account of its importance I quote it at length.

Case of Chronic Fibrous Myocarditis and Calcification of the Coronary Arteries, with severe pain in the left forearm.—Female, age 60, consulted me on 30th July, 1892, complaining of great irritation in the privates which comes on periodically and sets up a great pain that strikes into the left forearm. These attacks occur several times a day, and last from fifteen to twenty minutes. She feels great depression when the attack comes on. She is very short of breath, and there is slight swelling of the legs. There is considerable difficulty in making water. There is great thirst, fair digestion, and constipated bowels. The patient is pale and wan, and the face has a pained and anxious expression. The body is well nourished. The tongue is very dry. The urine has a specific gravity of 1030, contains a trace of sugar, but from enquiry the quantity of urine passed does not appear to be abundant. The site of the irritation is very definitely limited to the inner surface and skin edge of the vulva. The arteries are large and slightly atheromatous. The pulse is full, soft, quick, and regular. The left side of the chest is tender to pressure, but there is no cutaneous hyperæsthesia. There is a very tender spot on pressure over the 2nd left rib in the nipple line, and the upper 3rd dorsal vertebra is very tender. There is no defined apex beat. The area of the heart's dulness begins at the 3rd rib in the parasternal line, and extends transversely from the middle of the sternum outward to the left for four and a half inches. The heart sounds are sharp and clear in all the areas, but in the aortic area the second sound is markedly accentuated, resembling a drum-tap. There is marked pulsation of the internal jugular veins, and tracings of this pulsation taken at the same time as the radial pulse, show the pulsation to be due to the contraction of the right auricle.

No abnormality can be detected in any other organ. The patient was directed to diet herself carefully, and, for relief of the irritation, to inject hot water into the vagina by means of a Higginson's syringe during the attacks and systematically night and morning. She was asked also particularly to note the exact site of the pain when it recurred and its connection with the irritation in the vulva.

In ten days' time she returned. The pain she found had no relation with the irritation, but came on always with exertion. It was situated in the left forearm, and she defined very clearly the ulnar half of the forearm. Sometimes it was associated with a pain of less severity under the left breast. Under the treatment prescribed the irritation was greatly relieved, and the attacks had become much rarer.

The progress of the case was satisfactory so far as the urinary condition was concerned. The sugar after a few weeks' treatment disappeared from the urine, and the irritation also completely subsided. But the pain in the arm resisted all treatment. On one occasion she came to my consulting room in a terrible state of distress, and sat rocking the body to and fro, nursing her left forearm in her right hand. The onset of this paroxysm of pain had been induced in consequence of her having to hurry to catch the train by which she was coming to consult me. While she was under this attack I made a careful observation of the pulse and the site of pain. The pulse presented no change from its usual character, either to the touch or from the study of the sphygmogram. *The pain was exactly located in the ulnar half of the forearm*, no pain being complained of in any other part.

As I could give the patient no relief, she came into the Victoria Hospital under my care. She was put to bed, and not permitted to get up for three weeks. After the first week she had no attack, and after she was discharged she had them only very slightly and very rarely. I lost sight of her for a few months until the 9th of July, 1893, when she consulted me for an attack of diarrhœa. She said the old attacks of pain had never been so frequent since she had left the hospital, although occasionally they were still very severe, but were always limited to the left forearm. On examination during this visit I found only slight tenderness of the 1st dorsal vertebra and of the sub-cutaneous tissues in its neighbourhood. The heart presented no change except that the second sound, although still markedly accentuated, was not quite so clear, being somewhat muffled. She had her left forearm carefully wrapped in flannel, under the impression that it eased the attacks. There was no sensory abnormality demonstrable in the arm. On the 22nd I was asked to see her, as she was very ill. I saw her on the 24th. She was propped up in bed suffering from great dyspnœa, and the legs were greatly swollen. She died next morning.

Permission to examine the heart was obtained with difficulty, and only an incomplete *post-mortem* was therefore held. I sent the heart (which was empty and flabby) to Dr. Williamson, of Manchester, and his report is as follows :—

Heart weight, 10½ oz. Left ventricle slightly enlarged, very slight thickening of aortic valves. Pulmonary, mitral and tricuspid valves normal. No atheroma of the aorta. The muscle wall of the left ventricle is of normal thickness and consistency; but in the cut surface there are a number of small pale whitish patches of irregular size and shape. Both coronary arteries are markedly atheromatous and calcareous from their aortic origin down to their small sub-divisions. The main arteries and their chief branches can be dissected out as calcareous tubes. The cardiac muscle of the left ventricle when examined microscopically does *not* present any signs of fatty degeneration (osmic acid). Sections show patches of fibrous tissue amongst the muscle fibres.

The pathological diagnosis is *marked atheroma and calcification of the coronary arteries; chronic myocarditis (fibrous)*.

There can be little doubt that the cause of the pain in this case was situated in the heart. Putting aside the situation of the pain, all the other phenomena pointed to the heart as being the *origo mali;* as, for instance, the suddenness of the attack engendered by exertion, the dull, terrible agonising nature of the pain, and the great sense of oppression accompanying it. The peculiar face of the patient was to my mind also characteristic of the expression in patients who suffer much pain associated with disease of the heart. Concerning the situation of the pain, it was at last entirely situated in the arm. Occasionally at first she did complain of pain under the left breast, but in the few attacks which I have witnessed the pain was entirely located in the arm. And the area here was extremely definite in its limitations. When asked as to its situation she always stroked the ulnar border of the forearm from the elbow to the wrist. There was never any other disorder of sensation in this locality, neither of the skin nor deeper tissues.

The area, then, in which this pain was felt was in the region of cutaneous distribution of the 1st dorsal nerve. Considering the marked disease of the heart substance and of the coronary arteries, there seems little doubt but that the stimulation from them proceeded to the root of the 1st dorsal nerve, and the pain was then referred to its cutaneous distribution. Suppose now that the area of pain had corresponded to the area of distribution of the 3rd or 4th dorsal nerves in the chest—a circumstance far from unusual—would not one have said then that the pain was felt in the heart itself? And this view would have seemed all the more probable if there were complete absence of hyperæsthesia. It seems to me, in view of such a striking example as this case presents, that there is room for great doubt that the pain of an organ is necessarily situated in the organ simply because the pain may happen to be located in its neighbourhood. The pain in angina pectoris may extend in a direction the reverse of the course usually pursued, originating say at the wrist, and extending up the inside of the arm to the chest. Here it would seem that the irritation first affecting the root of the 1st dorsal spreads to neighbouring roots—characteristic of the manner in which the area of pain extends in diseases of other organs. Yet Walshe[1] looks upon such a manifestation as being an instance where "the brachial pain played the part of an aura."

Studying the subject from a purely clinical aspect, such are, in brief, some of the reasons that influence me in considering the pain of visceral disease to be of a referred character, always somatic, never splanchnic. I willingly admit I am not able to explain the source of all pains, and many patients complain of pain to me unexplainable. Yet with this view present in my mind, and with extended experience these cases become less numerous.

II.—*The Sensibility of the Visceral Tissues.*

A certain amount of light is thrown upon this subject when the sensitiveness to stimulation of the viscera is considered. The relative great bulk of the organs to the nerves that supply them and the varied functions these nerves subserve, suggest the idea that there can be but a very limited amount of sensibility.

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[1] *Loc. cit.*, p. 198.

In considering this subject it will be convenient to divide the tissues constituting the viscera into three, namely, the serous, muscular and glandular. This division will be useful in considering not only the sensitiveness of the tissues to ordinary stimulation, but also in considering the sensory disorders that arise during affections of the individual organs. To the methods employed for ordinary stimulation (touching, pricking, pinching, &c.) it is well known that the viscera are insensitive. But still, painful sensations can be aroused, and the symptoms thus elicited help in clearing up a good many obscure phenomena.

(A) *Serous Membranes.*

For many years when tapping for pleurisy I have been accustomed to note the sensations elicited when the visceral pleura came in contact with the canula. Coughing has been set up, but never any pain or other sensory phenomena. In like manner on testing the parietal and visceral pleuras with more facility in cases of excision of the ribs, I have failed to elicit any painful sensation. Richerand[1] long ago noted the insensibility of the pericardium, which he exposed in removing a portion of a scirrhus pleura. The insensibility of the peritoneum has been frequently demonstrated in operations affecting the various abdominal and pelvic viscera.

(B) *Muscular Tissue.*

In hollow muscular organs pain can be elicited by stimulating the muscle to contract, as after giving a distending enema the great gut contracts producing a referred pain in the abdominal wall midway between the umbilicus and symphysis pubis. The uterus can be made to contract, with the production of the pain in a similar area. The pains may extend in both cases to other regions, but in no sense are they felt in the contracting organ. But to any stimulation, save that which gives rise to powerful contraction, the muscular is as insensitive as any of the other tissues.

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[1] Richerand A., "Elements of Physiology." Edited by James Copland. 2nd edition, 1829, p. 613.

The insensitiveness is still marked when the parts are inflamed. In a patient from whom a large piece of gangrenous intestine had been removed, the exposed gut a few days after was deeply congested and swollen. But stimulation of the peritoneal and mucous surfaces and intervening tissues was not perceived by the patient.

Seeing that it is the muscular tissues that evolve pain most readily, it will be best to study shortly the sensory phenomena associated with the hollow muscular viscera. No one doubts but that the pain felt during painful peristalsis of the bowel, or during uterine contractions, is produced by the muscular tissue. The pain so produced may be still felt after the contraction has ceased as a feeling of soreness, and hyper-æsthesia can frequently be demonstrated. It is a curious fact that the pains, and other sensory disorders that are most severely felt by patients, are those that arise from affections of the hollow muscular organs. I do not mean to imply that spasm of the muscle is necessary to the production of pain. In angina pectoris, this is manifestly not the case, notwithstanding many opinions to the contrary. But the pains, for instance, that are the most marked and can be referred with most certainty to the organ affected, are those of the heart, of the stomach, small and great intestine, and of the uterus. The pains, likewise, of gall stone and renal calculus are in all likelihood essentially those of hollow muscular organs. The calculus lodged in the substance of liver and kidney gives rise to no sensory symptom, but when it invades the hollow viscera connected with them then the pain is aroused. I have had frequent occasion to observe the persistence of pain, and hyperæsthesia after the expulsion outside the body of gall stone and renal calculus. In view of the possibility of these symptoms, and those also manifested so frequently in stomach affections, being associated with and probably due to spasm of muscular tissue, the following remark by Beaumont in his observation upon Alexis St. Martin is of interest.[1] "If the bulb of the thermometer be suffered to be drawn down to the pyloric extremity, and detained there for a short time, or if the experiment be repeated too frequently, it causes severe distress, and a sensation like cramp or spasm, which ceases on withdrawing the tube ; but leaves a sense of soreness and tenderness at the pit of the stomach." I have little doubt in my own mind, but that the tenderness and soreness here described, was in reality due to a condition of hyperæsthesia of the skin or subjacent tissues. I usually find these phenomena when patients complain of such feelings in the epigastrium. In a case of lax abdominal walls Macdonnell[1] was able to palpate the under surface of the heart, and pressure upon the auricles gave rise to symptoms resembling angina pectoris. Without seeking, therefore, to insist that the pain in visceral disease is due in all cases to muscular spasm, I would seek to point out, first, that pain and other sensory phenomena are frequently associated with hollow muscular organs; second, that symptoms exactly analogous to these produced by disease can be originated by artificial stimulation of the muscles of hollow organs.

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[1] Beaumont, W., "Experiments and Observations on the Gastric Juice, &c." Edited by A. Combe, Edinburgh, 1838, p. 105.

(C) *Parenchymatous Tissues.*

Diseases of the parenchyma of an organ are not associated with very definite sensory symptoms. In fact, I have more than a suspicion that there are none. I have carefully enquired into the history, and watched the progress of many cases of acute nephritis from the onset, and yet have been unable to find any sensory symptom associated therewith. The ravages of consumption are notoriously painless, unless the pleura be involved. The pains of pneumonia are those observed in pleurisy.

A certain amount of pain can be elicited by pressure on a number of the organs (kidney, testicle, ovary, &c.). But here, so far as I have been able to make out, the pain is a referred pain.

When pain occurs associated with disease of an organ, it can frequently be demonstrated that there is an extension to the serous covering, as in the pleurisy of pneumonias. I have had frequent opportunities lately of studying the pains associated with great enlargement of the liver from disease of the heart. In such cases pulsation of the organ is a frequent symptom, and in the course of an enquiry into the nature of this pulsation, I noted the frequent occurrence of pain over the distended organ. So far as I could make out it was evidently on the surface of the organ, and in one case where the pain and tenderness were very marked, there were found in the *post-mortem* examination numerous adhesions between the anterior surface of the liver and the abdominal wall.

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[1] Macdonnell, H., "Cardiograms from the Human Heart," *The Practitioner*, vol. xliv., 1890, p. 178.

I have already referred to the pains associated evidently with the ducts of certain organs (bile ducts, ureters), and it is a fact worth noting that diseases of a ductless gland like the spleen appears to be unaccompanied by any sensory symptoms. One great obstacle in regard to this theory of the painlessness of parenchymatous disease, is the pain associated with orchitis, unless it be due to the swelling pressing on the more sensitive tissues external to the organ. Otherwise, why inflammation of this organ should produce such pain, and inflammation of the kidney should be painless, I do not understand. I am conscious that many very capable physicians affirm with great assurance that there are pains distinctly associated with disease of the parenchyma of organs. But all I can say is that looking at the matter dispassionately I have yet found no distinct evidence of such a thing, and I do not feel justified in ascribing a pain, whose origin I do not know, to an obscure organ whose sensory manifestations I do not understand.

III.—The Character and Distribution of the Sensory Symptoms in Inflammation of Serous Membranes.

When all the sensory symptoms that can be elicited from a series of cases of inflammation of the pericardium or pleura, are grouped together, it seems very difficult to find a satisfactory explanation for them.

The assumption that the pain is local and due to stimulation of the peripheral nerves, the view most commonly held and supported by Ross and Head, as distinct from a reflex or associated pain, is not sufficient. A great variety of these sensory phenomena may present themselves in cases that are otherwise apparently identical so far as our means of physical diagnosis are concerned. In looking over my notes of six cases of pericarditis I found that two presented no sensory phenomena, that a third had a slight pain referred to the upper part of the præcordial region, that a fourth had pain for a few hours of agonising severity across the middle of the sternum. I was summoned to see this patient between three and four in the morning, and the symptoms resembled those of an attack of angina pectoris—the ashen hue of the patient's face, the fear of impending death, and the agonising pain. The pulse was quiet and regular, and the only abnormality was a to and fro murmur over the area of the heart's dulness. After the subcutaneous injection of a large dose of morphia the pain abated. Later in the morning the pain had entirely gone, and the friction murmur had nearly disappeared, and in a couple of days had entirely disappeared. Since then (three years ago) the patient has had no recurrence. The last two cases presented somewhat similar phenomena; one of them only I will detail.

Case of Pericarditis with marked sensory disorders.—Male, aged 23, thin and meagre, had been under my care occasionally for nine months, chiefly on account of obscure abdominal symptoms (pain and diarrhœa). I had kept him under observation chiefly on account of marked pulsation in the veins of the neck and sometimes in those of the chest wall. The venous pulse was of the auricular type, *i.e.*, the main wave of the pulse was due to the auricular systole. There was also a marked extension of the heart's dulness to the right of the middle line for two inches. He had occasional attacks of violent palpitation in the night. On the 18th March, 1893, he left his work at eleven in the morning, feeling tired and exhausted. On the 21st he was seized with a pain of great severity in the chest, with difficulty in breathing. I saw him on that day and found him sitting up in bed, holding his body very rigid and breathing with evident difficulty, and on his face an expression indicative of great suffering. The pain struck from the præcordial region round to the left shoulder blade (so that he could not lift his left arm) and down the inside of the left arm and ulnar part of the forearm to the little finger. Any movement of the body caused pain, and he was too terrified to take a deep breath. There was extreme tenderness of the skin and sub-cutaneous tissues over the whole front of the chest as far out on both sides as the nipple line. Over the 3rd left costal cartilage and adjacent portion of the rib, this tenderness to pressure was excessively acute. Careful note was made that the tenderness referred entirely to the tissues in the external body wall, and that the method to elicit this tenderness did not affect the heart substance. The pectoral muscles, and the structures forming the anterior and posterior walls of both axilla were also very tender. There was also slight tenderness to pressure of the left sterno-mastoid and trapezius muscles, one point over the trapezius immediately above the middle of the scapular spine being especially tender. The muscles over the back of the scapula were also very tender. On auscultation over the heart, a loud to and fro friction murmur could be heard over the whole heart. There was no murmur accompanying the heart sounds. The pulse was quick, small and regular, 110 per minute. The temperature was 103° F.

For three days these symptoms remained practically unaltered, the pain only yielding to large doses of opium. During the time he was so ill I asked two of my colleagues to visit him, who verified the diagnosis and the above description of the symptoms.

(In view of the resemblance of the symptoms in these cases to those of angina pectoris associated with disease of the myocardium, the suggestion has occurred to me that it is possible that the pain, even of pericarditis, may be due to the invasion of the muscular tissue of the heart by the inflammation).

In pleurisy similar phenomena are sometimes met with. Thus in a case recently under observation, the onset of well-marked dry pleurisy gave rise to no pain whatever. The friction was at first limited to the right base, and attention was only directed to the condition by a sudden rise in temperature. The friction extended after a couple of days round to the front, and could be heard over the lower third of the chest wall; at the same time symptoms of pain and tenderness on pressure were developed. There was no cutaneous hyperæsthesia, and the tenderness was well-marked on percussing the chest. This tenderness extended higher than the friction sounds, and it is quite possible that the pleurisy extended over a greater area than that over which the friction was heard. There was no tenderness, however, of the chest wall behind the posterior axillary line, although the friction sounds were heard over this area in the lower part of the chest. But the interesting point in this case was the tenderness of the muscles forming the anterior and posterior walls of the right axilla. Thus, on gently grasping the skin no increased sensation was produced, but on grasping as gently the muscles, then the patient winced from the severity of the pain. On the other hand, no symptom of pain was elicited on applying the same test on the left side. Without entering minutely into the particular nerves here manifesting this abnormal sensitiveness of the axillary walls, it may be pointed out that none of them came within the region of the inflamed pleura.

Hilton has remarked upon the sensitiveness of the skin in pleurisy, and evidently considers it as due to reflex stimulation of the nerve root, comparing it to the pain felt on the inner side of the knee, in disease of the hip joint. In fact, Hilton bases an elaborate argument upon the intimate association of the nerves supplying a joint, the muscles acting upon the joint and the skin covering the muscles, and looks upon the great serous cavities as being analogous to the joints of the extremities. I have frequently observed well-marked hyperæsthesia in cases of pleurisy, and that in a situation that could not be explained by the pleurisy affecting the branch of a nerve and so producing the hyperæsthesia.

Case of Basal Pleurisy with abdominal Hyperæsthesia.—Female, aged 31, complains of pain in her left side, especially on taking a deep breath. The patient is thin, but fairly nourished. She lies in bed slightly propped up. The temperature is 100° F. The respirations are quick (32 per minute) and shallow, and each respiration is suddenly checked. The pulse is 104 per minute, small and regular. There is great pain and soreness in the left hypochondrium. An area of hyperæsthesia can be readily mapped out, extending as a band from near the middle line in front round the left side of the abdomen to the spinal column. The breadth of the band is about three inches. In the front the umbilicus is situated opposite the middle of the band, and at the sides it crosses the tips of the floating ribs, and terminates behind opposite the 12th dorsal and 1st lumbar vertebræ. The edges of the band are not well defined. The chest is resonant on percussion, and on auscultation the only abnormality is a small area over which friction is very distinct at the base of the left lung behind. These symptoms persisted for a couple of days, then disappeared together, that is to say, on the third day the temperature was normal, the respirations quiet and free from pain, the hyper-æsthesia was gone, and no friction could be detected.

I have, in a great number of cases of pneumonia, found a patch of hyperæsthesia over the area in the abdomen where pain is so frequently complained of in such patients. This abdominal pain of pneumonia is a well-known symptom, but its association with hyperæsthesia has not been sufficiently recognised, although Valsalva mentions it in a case quoted by Morgagni.[1] I have rarely found it quite so extensive as in the above case, and I have always viewed it as being associated with diaphragmatic pleurisy, a view that finds support in the symptoms of such cases as the one just recorded.

A shoulder pain associated with disease of the abdominal organs has long been recognised, and has been the object of particular study by Peter,[2] who associated it with the phrenic nerve, irritation being conveyed by this nerve to the roots of the cervical nerves, and the pain referred to their cutaneous distribution—a view that receives the support of Ross. In several cases of basal pneumonia and of pleurisy limited to the lower margin of the lungs I have found not only this shoulder pain, but also a distinct field of hyperæsthesia on the shoulder. In such cases I have attributed the shoulder pain and hyperæsthesia to the accompanying diaphragmatic pleurisy (although it may be due to the vagus terminations being involved by the inflammation of the lung). It is difficult to account for these phenomena satisfactorily. Hilton describes the parietal pleura as being supplied by intercostal nerves. If so, they must be very scant. Some years ago I made a careful dissection of several intercostal nerves, first following the trunk of the nerve and tracing each twig to its destination, but I could not find a single filament going to the pleura.

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[1] Morgagni, H., "De sedibus et causis morborum," book ii., letter xx., article 30.

[2] Peter M., "Névralgie diaphragmatique," &c. *Archives Générale de Médecine*, June 17, 1871.

If the suggestion be found tenable that the far-reaching pain and hyperæsthesia accompanying pericarditis is due to an extension of the inflammation to the myocardium, then the abdominal and shoulder pains of basal pleurisy may in like manner be due to an invasion of the muscular tissue of the diaphragm by the inflammation.

Of the sensory phenomena associated with disease of the peritoneum I have very little experience, and I am unable to give any definite results. I may say that since I recognised the hyperæsthesia associated with diseases of definite organs, I have had fewer cases of "peritonitis"—for sometimes the hyperæsthesia in, say, a case of pyelitis with retention of pus, gives rise to symptoms resembling the descriptions of extreme tenderness of the abdomen in cases of peritonitis.

When, however, the various sensory disorders associated with the diseases of the serous membranes are considered, there is some difficulty in getting a clear conception of how they are produced. At all events, the statement that "affections of the serous cavities of the body do not cause referred pain or cutaneous tenderness, but produce local pain which follows the lines of peripheral nerves, and is associated with deep tenderness over the affected point only" (Head) is not sufficient explanation for the conditions present in such cases as I have quoted above.

If this view of the absence of pain in the viscera be substantiated, it would lead one to surmise that the great organ of sensation, the skin, is altogether a later development in animal life, having for its function the protection of these viscera necessary to the maintenance of organic life. The sympathetic nerves would therefore have functions apart, and distinct from those of the spinal nerves. This independence of the sympathetic nervous system has received a very elaborate exposition by Davey.[1] Support for this conjecture is found in the recent observations of Paterson,[1] who maintains that the sympathetic nervous system is developed independently in the mesoblast, and does not arise as an outgrowth from the cerebro-spinal system. The elaboration of this view necessarily leads to the discussion of matters pertaining to morphology and development—subjects which I am not qualified to discuss.

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[1] Davey, J. G., "The Ganglionic Nervous System," London, 1858.

IV.—The relationship between the Areas of Cutaneous Hyperæsthesia associated with Visceral Disease, and the Areas of Cutaneous Distribution of Spinal Nerves.

In my earlier observations I had come to the conclusion that when there was a limited field of hyperæsthesia in any given case of visceral disease, it occupied a situation in some part of the cutaneous field of distribution of a spinal nerve, whose root was in intimate association with the root of the sympathetic nerves that supplied the affected organ. When the hyperæsthesia became more extensive, it spread in a somewhat diffuse manner into fields occupied by spinal nerves, whose roots are in close relation with the one originally affected. While, therefore, there was a continuity in the skin area affected, that continuity always occurred in a definite direction, namely, in accordance with the distribution of the spinal nerves whose roots are in close association. Thus, the hyperæsthesia in heart affections beginning in the 4th dorsal nerve distribution might spread up the chest, but instead of passing over the clavicle into the field of supply of the 4th cervical it passes down the arm along the fields of supply of the 3rd and 2nd dorsal. In spreading thus it did not necessarily affect the whole area of supply of one nerve root. Thus, one portion of the field of supply of the 4th dorsal nerve might be hyperæsthetic. As this area increased in size, it did not first spread over the whole field of supply of the 4th nerve, then invade the field of supply of the 3rd. Occasionally it might do so, but more frequently if the area was limited, as it usually is, to the anterior surface of the chest;

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[1] Paterson, A. M., "Development of the Sympathetic Nervous System in Mammals," *Philosop. Trans.*, 1890.

this area would extend into the armpit and down the arm without involving the posterior surface of the chest. There does not appear to be any law regulating this spreading. Thus, in four cases of gastric ulcer at present under observation, in all of whom there has been lately vomiting of blood, an examination shortly after the hæmatemesis revealed the following conditions relating to the sensory symptoms. In two there are absolutely no sensory symptoms. In one there is well-marked hyperæsthesia in the epigastrium shading off into a less definite field of hyperæsthesia that extends from the level of the umbilicus round to the left to the spinal column, and up over the thorax and down the inside of the left arm, and the ulnar half of the forearm to the little finger. In the fourth case the hyperæsthesia occupies the front of the abdomen, reaching on both sides as low as the groin, with a tongue-like projection into the front of the thighs, similar to that Head maps out as being characteristic of the distribution of the 12th dorsal nerve. On the right the hyperæsthesia reaches as high as the umbilicus, and on the left as high as the ensiform cartilage. It does not extend backwards on either side beyond the anterior axillary line. In this latter case there is remarkable extension of the area in which the uppermost epigastric reflex can be produced. In many persons the reflex contractions of the rectus abdominis muscle above the epigastrium can be observed to occur in three divisions corresponding doubtless to the divisions produced by the tendinous intersections. In this patient stimulation of the skin as high as the nipple frequently (and sometimes even the skin in the axilla) will provoke a contraction of the upper portion of the rectus on the left side. The skin here is not hyperæsthetic in the slightest degree.

V.—Overlapping of Sensory Fields.

In very few of the cases could the field of hyperæsthesia be delimited with certainty. Parts at the border would be hyperæsthetic at one time, and when one would return there would be some doubt, or a distinct assertion that the part was not hyperæsthetic. In the centre of any limited area the hyperæsthesia was more acute. Under these circumstances I did not think that the hyperæsthesia associated with visceral disease afforded a sufficiently accurate clue to the delimitation of the areas supplied by individual nerves, and I looked upon this variation in the intensity of the hyperæsthesia as being due to an over-lapping in the distribution of neighbouring nerves. This view was strengthened by the fact that I had seen evidence of a slight extension of the hyperæsthesia into fields supplied by nerves whose roots were not in neighbouring association. I considered also that I had obtained evidence of this overlapping from the study of the eruption in cases of herpes zoster.[1] When Dr. Head published his extremely exact delimitation of the fields, I imagined that I had not been sufficiently accurate in delimiting my fields. I therefore made another series of careful observations, and although in a few cases I appeared to get results that partly supported his views, I must confess to having utterly failed in the great majority of cases. Even in the study of a few cases of herpes zoster I could not get them to fit into any one of his fields. To demonstrate these several points I will quote the following three cases that merit publication for other reasons.

Case of Herpes Zoster without eruption.—Male, aged 53, complains of pain on the top of the right shoulder extending down the arm to the thumb. The pain began about three weeks ago, and has continued ever since. Sometimes it comes on in severe paroxysms, accompanied by a disagreeable smarting sensation. He can scarcely bear his clothes to touch his shoulder, and he can't wear his braces over the right shoulder because of the pain it causes. The paroxysms of pain have prevented him sleeping of late, and in consequence he is getting tired and weak. The patient is a big, stout man of healthy appearance. When stripped there is nothing abnormal to be observed in the skin of the right shoulder or arm. On testing the sensation, the skin on the top of the shoulder is markedly hyperæsthetic. The hyperæsthesia occupies a very characteristic situation, which can be easily mapped out, as it is so acute the patient has no hesitation in noting when the stimulation passes from the healthy skin to the abnormally sensitive. It begins abruptly in the neck about the level of the 3rd cervical vertebra, and extends round the neck at that level to the middle line in front. It extends from hence over the top of the shoulder down the outer surface of the arm, and radial half of the forearm to the thumb. The posterior border of this area extends from the 3rd to the 6th cervical vertebra, then slants outwards at the level of the spine of the scapula to the posterior surface of the shoulder, then down the back of the arm past the side of the olecranon into the forearm to the thumb. The anterior border descends in the middle line to the level of the clavicle where it slants slightly outwards to the level of the third rib. It then runs transversely out to the front of the shoulder, where it descends in the front of the arm and forearm to the thumb (fig. 1). This area, although so distinct, does not evince the hyperæsthesia with equal intensity in all parts. Thus in testing round the arm, when passing from the healthy skin, the hyperæsthesia at the borders is not so acute as towards the centre of the affected area. The under surface of the thumb is slightly anæsthetic. The same results are obtained on testing by heat.

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[1] Mackenzie, J., "Herpes Zoster and the Limb Plexuses of Nerves," *Journal of Pathology and Bacteriology*, vol. i., p. 332.

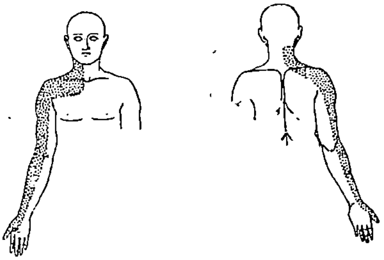

FIG. 1.

Careful enquiry was made into the condition of all the other organs, but with the exception of a slightly furred and fissured tongue no abnormality could be detected.

The patient called to see me at intervals of five or six days, and each time I mapped out the hyperæsthesia, the results always being practically the same. In a month from the date of his first visit the hyperæsthesia had gone, and with it to a great extent the pain. For several weeks later the pain used to recur in slight paroxysms.

I have assumed this case to be one of herpes zoster, although in the strictest sense it has no right to the term herpes or zoster. But that it is essentially the same disease I have little doubt. The description of the pain—the paroxysms, its wearing character preventing sleep, and its duration are exactly the symptoms described by patients who suffer from herpes. Although I have never read of any account of herpes zoster without an eruption, I think that it may not be so very rare. Some years ago I saw in consultation a patient said to be suffering from some painful disease of the spine. In her there was a distinct area of hyperæsthesia following the distribution of the 4th dorsal nerve. The pains were characteristic, and located chiefly within the hyperæsthetic area, but radiating into the arm. I detected on the back two small vesicles, and on the strength of this, suggested the diagnosis of herpes zoster. The medical man in charge attributed the blisters to his applications, but on enquiry they were not such as would give rise to vesication. The future progress fully confirmed this view of the case. Lately I had a patient with severe pain in her side, and a distinct band of hyperæsthesia embracing one half of the body. I was a few days in doubt what might be the nature of the complaint until it was disclosed by the appearance of a few vesicles in the epigastrium, the remainder of the hyperæsthetic field remaining quite free. Dr. Head has likewise noted similar cases, and von Bärensprung,[1] has described the intervals of skin between the eruption as being hyperæsthetic. So that it would appear that an eruption is not a necessary symptom in herpes zoster.

On the question of differential diagnosis the only alternatives that occur to me, are that this case was an irritation of nerve roots due either to disease of the vertebræ or to some visceral lesion. That it was not due to a disease of the bones is evident from the progress of the case. There was never any spinal tenderness. I made, as I have said; enquiry into the condition of all the other organs in the head, throat, thorax, and abdomen, but could find no clue. The vagus and phrenic roots being in association with the cervical nerves led me to make careful examination of the organs supplied by them, but the quest was vain.

For these reasons I consider the case as being due to an affection of the roots of the 3rd, 4th, 5th, and 6th cervical nerves, and to be of the same nature as that which gives rise to the disease known as herpes zoster. And the case illustrates in a very definite manner the cutaneous area supplied by these nerves. The relations of this area to neighbouring areas are of considerable importance, and will be referred to later.

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[1] Von Bärensprung "Die Gürtelkrankheit," *Annalen des Charité-Krankenhauses*, Band ix., Heft. 2, 1861, p. 103.

Herpes Zoster of the 3rd dorsal nerve with Hyperæsthesia.—Female, aged 13, has been under my care for over a twelvemonth for mitral disease, with symptoms of cardiac failure. On the 10th August, 1893, I was consulted because of an eruption that had broken out on the right chest. There was very little pain except on the inner side of the right upper arm. The eruption consisted of clusters of vesicles disposed as an interrupted band round the upper part of the chest and down the inside of the forearm. In front the clusters were continuous at their borders and represented a band about two inches broad. The most internal portion was situated over the sternum opposite the 4th costal cartilage. A few vesicles trespassed across the middle line for quarter an inch. From here the band extended outward and slightly upward, crossing over the 3rd rib to the front of the shoulder. A few scattered vesicles extended as high as the 2nd rib, and as low as the nipple—some being situated on the nipple. Behind the eruption extended as a somewhat broader band from near the spinal column, the centre being opposite the 2nd dorsal vertebra, outward and slightly downward over the scapular spine to the posterior axillary fold. On the right arm the eruption occupied the inner surface, two streaks occurring on the anterior and posterior axillary folds, and other two streaks occupying the anterior and posterior surfaces of the inner aspect about the lower third of the upper arm. Surrounding the area of eruption was a wide field of hyperæsthesia. In front it was as high as the second rib and crossed the middle line for at least one inch. Behind it ascended as high as the level of the 7th cervical vertebra and also crossed the middle line for a very short distance. It descended the side of the chest in an irregular manner as low as the 10th rib. It also descended the arm embracing the area of eruption. The interrupted line in fig. 2 shows the hyperæsthetic area.

The hyperæsthesia in this case extends somewhat diffusely, yet in a definite manner. The irritation from the diseased root that produces the eruption spreads to neighbouring nerve roots. But in doing so it evidently does not necessarily affect the whole of one root before it passes to the next. Just as was pointed out in relation to the hyperæsthesia associated with visceral disease, contiguous fields are affected, without of necessity the whole area of supply of individual nerve roots being affected. The extension of the hyperæsthesia likewise takes place in a definite direction, not passing for instance over the clavicles into the field of supply of a distant nerve root, but descending on the arm.

A comparison of these two cases also shows a distinct overlapping of nerves. While it is difficult to prove this overlapping by the study of areas supplied by neighbouring roots, the opportunity is readily afforded when contiguous areas of skin are supplied by nerves whose roots are wide apart.

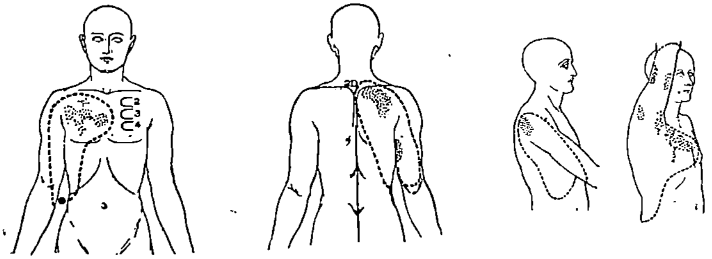

Fig. 2.

The interrupted line embraces the hyperæsthetic area.

Thus the skin of the upper part of the chest is supplied by the 4th cervical that descends over the clavicle as far as the 3rd rib in fig. 1, while the area supplied by the upper dorsal nerves ascends as high as the 2nd rib, as in fig. 2. This overlapping can also be seen at the back in these figures. This same spreading of the nerve supply of one field into that of another is also demonstrable in the crossing of the hyperæsthesia to the opposite side in fig. 2. The eruption has also crossed for a very short distance, and this was more unmistakably demonstrated in fig. 3.

I have been unable to find in my cases of herpes that accuracy of delimitation that Dr. Head describes. If the eruption in fig. 2 be studied, it will be found to occupy a fairly distinct field. The bands on the front and the back of the chest represent a fairly distinct continuity. But they will be found to correspond to none of the single areas described by Dr. Head. As a matter of fact, the eruption trespassed into the areas that he reserves for the 1st, 2nd, 3rd, 4th, and even 5th dorsal nerves. Yet the continuity of the eruption suggests to me very strongly the idea, that there is but one nerve root affected here—in all probability the 3rd. In fact, I look upon it as being a more complete delimitation of the same field that his case 14, fig. 13, imperfectly represents. This is, I think, strikingly evident in the posterior band, where it cuts by a straight band the wavy contours that he depicts. In making my notes of this case I carefully copied the outlines of Dr. Head's fig. 13, in order that the comparison might be as true as possible. These copies made by me were repeatedly verified.

As a further illustration of the disagreement of my observations with those of Dr. Head in this exact delimitation of the field of supply by individual nerve roots, I give the notes of another case of herpes zoster under the care of my friend Dr. Harwood, to whom I am indebted for the opportunity of seeing the case, and for his kindness in verifying the particulars.

Dr. Head describes the lower border of the field of supply of the 12th dorsal nerve as sending two tongue-like projections down, one on the front of the thigh, and the other over the side of the haunch. These depressions dovetail into the field of distribution of the first lumbar. In a few cases I have certainly found the area of hyperæsthesia associated with disease of certain organs assume this shape. But that it represents the exact limits of the 12th nerve only is not supported by the symptoms in the following case.

Case of Herpes Zoster of first lumbar nerve.—Female, aged 50, suffers from attacks of excruciating pain round the right hip, where an eruption has broken out. The eruption extends from the vertebral column at the lower lumbar region round the right hip to the front and inner surface of the thigh in a band from four to five inches in breadth. Its superior border is extremely well defined, slanting in almost a straight line from the vertebral column at the level of the iliac crest, round the side of the ilium midway between the crest and great trochanter, to the front of the thigh a couple of inches below Poupart's ligament, and almost parallel with it to the inner surface of the thigh. The lower border is not so clearly defined. It leaves the vertebral column at the level of the upper part of the sacrum, passes round over the ilium, and on the front of the thigh it is more irregular, sending scattered patches as low as the lower third of the thigh. The eruption distinctly crosses the middle line behind for at least the distance of one inch (fig. 3).

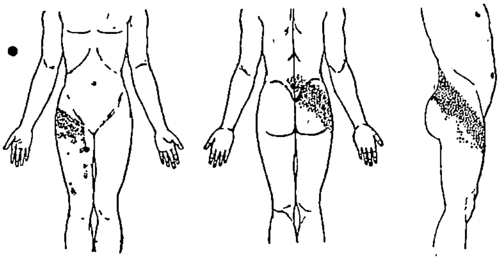

FIG. 3.

Here we have an eruption pursuing a fairly defined course, the upper limits of which slant in almost a straight line from the vertebral column to the inner surface of the thigh. Even if one assumes that part of the 12th dorsal root is involved in this case, it is surely too much to expect that the disease has picked out the portions of the roots affected, whose cutaneous distribution exactly fills up the gaps formed by the irregular outline of the 1st lumbar. Here again, there is undoubted crossing of the eruption to the left of the middle line, a further proof of the overlapping of the nerves.

From the study of cases of herpes zoster, and of the hyperæsthetic areas associated with visceral disease, I have come to the conclusion that there is distinct overlapping of the fields of cutaneous supply of individual nerve roots, of pathic, thermic, and trophic fibres, as well as of those of ordinary sensation. In this respect the results are in harmony with those arrived at by Sherrington from observation of isolated areas producing reflex contraction, with those obtained by Paterson from dissection of fibres from individual nerve roots, and with the observations of Langley on the pilomotor nerves.

VI.—Other Reflex Phenomena in Visceral Disease.

Besides the symptoms elicited from stimulation of the sensory nerve roots, a great number of other phenomena may arise from the stimulation affecting roots of nerves with different functions. If we consider that a stimulation arises in an organ and is transmitted by afferent nerves to a more central situation, and if this stimulation be of sufficient force or of the proper quality to affect certain nerve roots with which it comes in contact, then, according to the function of the nerve root, there will arise phenomena peculiar to the organ stimulated. Thus, if it be a sensory nerve root, there will be an exaltation of its function, giving rise to exaggeration of its peculiar property, or, it may be, causing a lowering of its functional activity, as when anæsthesia occurs. If the nerve root so stimulated be motor in function, then there may arise spasmodic contraction, or even loss of power in definite muscle groups, slowing of the beat or palpitation in the case of the heart. If the nerve root be secretory in function, there may arise an increased activity of the gland whose root is stimulated. In one remarkable case of angina pectoris I had under observation, the attack used to radiate characteristically from the chest to the left arm, and up into the neck. At the same time there used to be a striking and excessive discharge of saliva from the left side of the mouth. In another case of angina pectoris with aortic aneurism, where the pain was wont to radiate down the inside of the left arm, in this area, after a series of attacks, there was gradually developed a streak of ecchymosis, six inches in length. No cause could be assigned for its appearance, and I assumed it to be an affection of the trophic fibres. Hilton refers to certain trophic changes in the skin from disease of the teeth, and I have seen a case of ulceration of the abdominal skin after an attack of gall-stone colic, but as mustard had been used locally, I could not satisfy myself that it might not have something to do with the ulceration. Did we comprehend fully the manner in which this stimulation arising in a diseased organ spreads, we might be able more clearly to understand the meaning of many obscure cases of perverted functional activity of the various organs in the body.

VII.—Effects of Stimulation of the Skin upon the Viscera.

A certain amount of light is thrown upon the subject of the relationship between the cutaneous and the visceral innervation by the study of the effects of stimulation of the skin upon the viscera. The subject from this aspect is akin to that of the cutaneous reflexes, and doubtless lies at the foundation of the effects of treatment by counter-irritation, &c. There are certain well-known effects produced in the viscera by stimulation of the skin, and these are frequently taken advantage of in practice, *e.g.*, the counter-irritation in the pit of the stomach to allay vomiting, and the application of cold to the abdomen to cause firm uterine contraction. A very interesting light is occasionally shed upon this subject by the results of operations, as in the fact that after operations involving the skin of the perineum (for hæmorrhoids and for repair of torn perineum), not unfrequently retention of urine follows. Hilton had noticed that soothing applications to the perineum were beneficial in certain forms of retention of urine. Berry Hart[1] has recently noted a very striking instance of this relation between perineal skin and the muscles involved in the urinary reflex. Remarking the frequency with which retention of urine followed operations on the perineum, he determined to avoid the skin in his sutures. In a series of cases in which this plan was carried out the result was eminently satisfactory, as retention of urine no longer occurred.

The interest from the point of view with which I am dealing here is, that evidently the sacral nerves supplying the perineum are in association with the muscles of the bladder, and not of the mucous membrane alone as Head inclines to suggest.

VIII.—Practical value of recognising the Sensory Symptoms produced by Visceral Disease.

Treating the subject in this somewhat desultory fashion, it is difficult to convey properly the help that is afforded by enquiring carefully into these sensory symptoms in one's every day work. I might be permitted to single out a few instances for the purpose of illustrating the value of those symptoms.

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[1] Hart, D. B., "Perineal suture in relation to retention of urine after labour," *Edinburgh Hospital Reports*, vol. i., p. 587.

I have elsewhere called attention to the particular value of the seat of pain in intestinal obstruction. I do not see a sufficient number of cases to enable me to speak dogmatically on the subject, but in those I have observed, the results have been as striking as they were instructive. The pain in any given case of obstruction is due to the peristalsis of the bowel above the part obstructed. If, then, the pain is situated about the umbilicus, radiating more or less to one side, never descending lower, then one can infer that the point of obstruction is above the great intestine. If, however, the pain be felt as low as midway between the umbilicus and symphysis pubis, then some portion of the great gut is above the point of obstruction. In the latter cases the pain may arise in the umbilical (or even epigastric area) and gradually descend. It is necessary to see the patient when the painful peristalsis is upon him and to enquire carefully of him during the pain, as otherwise the information is too vague to be of service.

The observations that these hints are based upon, are from a dozen cases of hernia of the small intestines, and half-a-dozen cases of obstruction in some portion of the great intestine. The diagnosis was verified either by operation or *post-mortem* examination. These are certainly far too few observations to base an absolute statement upon, but they are of value as suggesting a help in the elucidation of a notoriously difficult point of diagnosis.

The sensory phenomena due to visceral disease may mislead very skilled observers. During my earlier observations on this subject I was called in consultation to see a man in failing health, in whom there was great pain round the left half of the body. There were remarkably few symptoms, besides the pain, and I found a hyperæsthetic area extending from the epigastrium round the left half of the body to the 6th and 7th dorsal vertebræ, which were extremely tender to pressure. I was puzzled with the case, and could only suggest that there was something wrong with the stomach. The patient receiving no benefit from my suggestions, consulted a skilled and deservedly eminent neurologist, who, on the strength of the tender vertebræ and hyperæsthesia, diagnosed disease of the vertebræ, and recommended the encasing of the patient in a plaster of Paris jacket. This was done, with no relief. In course of time the patient died, and his medical attendant wrote me saying that he had had a *post-mortem* examination, and there was cancerous disease of the posterior wall of the stomach at the cardiac end, and the vertebral column appeared perfectly sound.

The following case illustrates how misleading the sensory phenomena may be in another way. I was called, in consultation, to see a young lady whom her medical attendant informed me was suffering from peritonitis. A year previous, while at a fashionable health resort, she was laid up for four months with peritonitis. The appearance of the patient was typical of that disease, the drawn face, the small quick pulse, and the flexed knees and high temperature. She shrank whenever we ventured to touch the abdomen. Accepting the case as one of peritonitis, in seeking a cause, I proceeded to palpate the region of the vermiform appendix. But the patient would not let me touch the skin, it was so painful. Testing with a pin-head, I found the skin in this region acutely hyperæsthetic, and I was able to map out an area roughly corresponding to the distribution of the 11th and 12th dorsal nerves on the right side. On careful exploration I found that the other regions of the abdomen were not really tender, her desire to protect herself from pain making her frightened of being touched anywhere. On enquiry into the state of the urine, I was informed it was all right, but on examination there was a small quantity of pus. On careful cross-questioning, a distinct history of severe spasms, prior to the last illness, was elicited. I therefore gave the provisional diagnosis of pyelitis. In a couple of days there was an abundant discharge of pus in the urine, the hyperæsthesia disappeared, the temperature fell, and the patient made a quick recovery from her anxious condition.

IX.—The Sensibility of the Subcutaneous Tissues.

A knowledge of the sensibility of the subcutaneous tissues of the external body wall would be of great value. Are the pains of tissues lying under the skin referred or local? It seems almost absurd to ask the question, yet there are a number of very curious facts elicited when the subject is enquired into—as the well-known pain at the knee from inflammation of the hip-joint, and the hyperæsthesia of the skin associated with joint disease. That the subcutaneous tissues can become acutely sensitive, has already been illustrated, and the occurrence of afferent nerve fibres in muscles has recently been put beyond doubt by the investigations of Sherrington. But when one comes to examine the matter it is very difficult to obtain satisfactory results. I have been in the habit for a long time of performing operations (after rendering the skin anæsthetic) without eliciting the slightest symptom of pain. Lately a number of hernias has been operated upon in the Victoria Hospital without an anæsthetic, and after the skin was cut no pain whatever was elicited. The last one, however, on which I operated, did not yield such conclusive results. In this case, when dissecting the tissues overlying the hernia, the patient occasionally called out. On testing the portion of tissue, the cutting of which had caused pain, I never could reproduce the sensation. After the hernia was reduced, I brought the deep parts together by a series of catgut sutures, until all the tissues, save the skin, were approximated. In such cases I do not suture the skin, but hold the edges together, and paint the line of incision with celloidin solution. This dries quickly and brings the skin edges firmly together. In this manner the whole of the tissues, from peritoneum to subcutaneous fat were systematically stitched, and there was not the slightest pain evoked.

In many ways the knowledge that subcutaneous tissues are practically insensitive to stitching is of great practical value. Thus in recent ruptures of the perineum, the torn surfaces can be brought at once accurately together, with practically no suffering on the part of the patient by avoiding the skin in suturing. When skin sutures are employed, the suffering is frequently so great that the perineum cannot be satisfactorily stitched without an anæsthetic.

These facts were matters of common knowledge in pre-chloroform days, and many surgeons of to-day are no doubt cognisant of them. In conversation with an aged surgeon, who had seen much practice before chloroform was introduced, he informed me that it was always understood that after the skin was cut no pain was suffered during an operation, unless occasionally when a nerve was cut.

THE SENSORY DISTRIBUTION OF SPINAL NERVES.

BY WILLIAM THORBURN, F.R.C.S.

OWING to its wide scope and the very special nature of the observations recorded in it, Dr. Head's paper[1] is one which it is impossible to discuss in detail, and subsequent research on his own lines can alone confirm, enlarge, or possibly in some respects correct his conclusions. My aim, therefore, in the following remarks will be rather to dwell upon those aspects of his work which come most closely into contact with some of the views which I have previously published; but in so doing I propose to take this opportunity of collating and arranging some of my own results, and of analysing the recent progress of our knowledge regarding the cutaneous sensory distribution of the spinal nerves. Before proceeding to do so we may, however, cast a glance at certain other important issues raised by Dr. Head's paper.

Briefly, Dr. Head may be taken to have demonstrated that:—

1. There are associated with various visceral diseases certain definite and constant areas of cutaneous tenderness, each area having a "maximal" region in which there is pain.
2. These maximal areas coincide with the areas which Mackenzie, Head himself, and to a less extent, other writers have shown to be the areas marked out by attacks of herpes zoster.

The conclusion drawn is that these areas represent the cutaneous distribution of the pain-appreciative or algetic fibres of each serial spinal segment, and as a corollary the distribution of the algetic fibres of each segment is thus deduced.

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[1] BRAIN, Part I., 1893.

It is to the data of this important corollary that I propose mainly to refer, noting how far they either confirm or conflict with the deductions which had previously been drawn from other methods of clinical research, and the results obtained by Prof. Sherrington, from his experiments on monkeys.

The first consideration will be that of the value of the *method* employed by Dr. Head, and here we are at once met by the objection that areas of pain and of tenderness are extremely difficult to determine, in view of the essentially subjective nature of the record. For this reason they are *per se* less trustworthy than are observations of anæsthesia, and every clinician knows the difficulty of accurately defining an anæsthetic area. To the observations upon herpes, however, this objection is quite inapplicable, that disease depicting its area of distribution by the most definitely objective and easily recorded of signs. And as the two series of observations—those of pain-areas and those of herpes-areas—coincide accurately, we can only conclude that Dr. Head has overcome the difficulties of his first method, and that his areas are undoubtedly the areas of distribution of certain serial sections of the nervous system, although it remains to be proved whether these "sections" are spinal segments, nerve roots, or other serial arrangements.

Contrasting Head's areas with those obtained by clinical observation of anæsthesia, or with Sherrington's experimentally determined areas, we are at once struck by their sharp delimitation, and by the total absence of fading margins, and consequently of overlapping of adjacent regions in the series. Now there cannot be a doubt that the anæsthetic area resulting from disease or injury of a nerve root has a fading margin; that is, that the cutaneous distributions of the spinal roots overlap, often to a large extent. This want of sharp limitation is seen in cases of section of nerve roots, and in most cases of injury to the spinal cord (in which the nerve roots are usually crushed at the region of injury); and it is especially well indicated in Sherrington's experiments. Head himself explains the sharp limitation of his areas by supposing that they are the representatives, not of the distribution of nerve roots, but of spinal segments, and he points out that visceral disease can only evoke cutaneous pain through the intermediary of such segments. This theory has thus an *a priori* basis of probability, and it is further in entire accord with what we know of the plexiform arrangements of the nervous system to suppose that, if there do exist such sharply defined spinal segments as are here assumed, each of them would have a connection with at any rate some adjoining nerve roots, and that thus the nerve root itself would represent not a pure segment but the mass of the fibres of its own segment *plus* some fibres from those above and below it. In this way we could at once explain the sharp definition of Head's segment-areas and the overlapping of the anæsthetic root-areas.

I believe also that there is some direct evidence that the spinal sensory segments are more sharply defined than are the root-areas. In most crushing lesions of the cord there is a coincident injury to one or more pairs of roots, which, arising above the level of the cord lesion, have not yet left the vertebral canal, and thus the upper level of the anæsthesia which we find in such a case represents not cord-anæsthesia but root-anæsthesia. In other cases the roots, being firmer and more resistant than the cord, escape injury, and our anæsthetic level is then truly that of cord-anæsthesia; and in penetrating wounds especially the cord may be divided without the roots being injured. Now I have for some time believed that when the roots were uninjured the anæsthetic line was more sharply defined than in other cases, *i.e.*, that "cord-anæsthesia," or "segmental anæsthesia" had a sharper boundary than "root-anæsthesia." This is a view to which I find it exceedingly difficult to apply the test of definite observations, because many considerations are required such as—Has hæmorrhage ploughed up the cord above the actual crush? Was the crush or other injury complete in the cord? And were the nerve roots really injured, or did they entirely escape? Owing to these difficulties we cannot draw certain conclusions from the notes of old cases which were not specially observed from this point of view, and I must therefore be contented with the expression of a personal opinion that purely cord lesions give a more sharply outlined anæsthesia than do lesions which involve also the spinal roots.

In conclusion, it may be added that although I am strongly inclined to accept Dr. Head's view that his areas represent the cutaneous distributions of spinal segments, there remains to be disproved another explanation of his facts, viz.: that his areas are simply the *maxima* of the over-lapping root-areas as usually recognised and as illustrated by Sherrington. Taking such a view we might readily suppose that the maximal region of the sensory distribution of the root would be that affected by tenderness in an irritative condition, or by a "trophic lesion," such as herpes. In fact we actually find within Head's tender areas other smaller maxima, wherein tenderness rises to the level of pain, and whence herpes spreads to the remainder of the area.

Having said so much upon the general aspects of Dr. Head's paper we may now pass to the special consideration of the distribution of the various spinal roots, contrasting the results arrived at by different methods and different observers. The method adopted by myself has been that usually resorted to by clinicians, and its basis rests on the following facts :—First, we are able at once to determine by anatomical observation the distribution of certain roots, which have no plexiform connections, and which can thus be traced from their origin to their termination; such nerves are the first lumbar (dividing into the ilio-hypogastric and ilio-inguinal), the fourth sacral, and most of the dorsal roots; the lower level of the distribution of the cervical plexus is also well defined. Secondly, we have cases of disease and injury affecting known and limited regions of the cord, or isolated roots; by subtracting from the anæsthetic areas in such cases the areas whose nerve supply is anatomically known we arrive at the areas of distribution of the remaining nerves. All that is therefore necessary is a sufficient supply of cases and a clear localisation of the lesion, and, for obvious reasons, injuries are best adapted to our purpose, these having, as a rule, at least a well-defined *upwards* limitation. Thirdly, the results thus obtained can be checked by observing the associated paralysis of muscles whose nerve root supply is known from other clinical observations or from such researches as those of Herringham, Paterson, Ferrier, and Yeo.

In the lumbar region, however, we are met by the difficulties that the whole of the nerves arise within a peculiarly small area, that the roots lie closely together in their long intra-spinal course, that the solidity of this region renders small and localised injuries infrequent, that the cases met with are few as compared with injuries of the upper part of the spinal cord, and, finally, that death rarely follows at a sufficiently early date to allow of satisfactory *post-mortem* observation of the primary lesion. For these reasons the lumbar roots are in some respects more difficult to trace than are those of the brachial plexus, but we may here derive much assistance from observation of lesions of the cauda equina, bearing in mind that, after a crush of the cauda, if certain nerves are paralysed (as regards either motion or sensation) whereas others are not, the injured nerves arise from lower roots than those which escape; and, when loss of function is partial only, such loss is more complete the lower the origin of the nerve root whose injury has given rise to it. The two last propositions, which I first published in "BRAIN" for 1888, are of general application; they have been of the greatest assistance to me in determining the distribution of lumbar and sacral roots, and have, I believe, been adopted by all subsequent writers. A more extended experience has now satisfied me that, in very rare cases, they may be departed from, and that they are therefore not absolutely reliable, but their general value has only been confirmed by subsequent cases, and Herter has since found an anatomical basis for the facts in the *post-mortem* observation that in a case of pressure on the entire cauda equina pathological changes were most marked in the lower roots.

Using the above methods and applying them to the study of a large number of cases of spinal injury, I was able to determine with some accuracy the sensory distribution of the roots of the brachial and lumbar plexuses, and the results thus obtained were published in various papers, collated in my "Surgery of the Spinal Cord" (1889) and somewhat amplified in the "Jacksonian Essay" of 1890, which, although unpublished, is accessible in the library of the Royal College of Surgeons. Since writing the latter I have seen no reason seriously to modify the conclusions which were then presented.

As regards the brachial plexus, "the fifth cervical root supplies the region overlying the deltoid muscle and the outer aspect of the arm and forearm as far as the styloid process of the radius, or base of the thumb, and the eighth cervical and first dorsal supply the little finger and inner side of the hand, forearm and arm, the remaining roots providing for the central parts of the limb on both anterior and posterior aspects. The exact lateral extent of the bands of skin thus supplied by each root can hardly be determined, and is indeed probably not a fixed quantity; the boundaries are usually ill-defined, and there can be little doubt that vicarious conduction of sensory impressions readily arises" ("Surgery of the Spinal Cord"). These sentences are illustrated by the following diagrams :—Fig. 1 shows the upper level of the anæsthesia,[1] which was sharply defined, in a complete crush between the fourth and fifth cervical vertebræ, in which the intrinsic muscles of the upper limbs were totally paralysed; it thus illustrates the well-known lower border of the distribution of the cervical plexus. In fig. 2 we have the very common type in which the fifth cervical root is spared, the injury implicating the sixth and subjacent roots. In such a case the deltoid, biceps, brachialis anticus and supinator longus muscles would escape paralysis. Of this type many cases are recorded, but, as will appear from the diagram, the exact extent of anæsthesia on the ball of the thumb is not determinable. (Cases 3, 4 and 8 of my book are of this type.) In two other cases we had the condition represented in fig. 3, the whole of the intrinsic muscles of the arm being paralysed on the right side, whereas the deltoid, biceps brachialis anticus and supinator longus escaped on the left; thus then the fifth cervical root is spared on the left side only, and the difference in the anæsthesia of the two arms is well indicated. In this case the area of retained sensation barely reached to the styloid process of the radius.[1]

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[1] It should be stated that in the majority of the cases quoted sensation was tested only by the point of a pin, so that we are dealing rather with sensations of pain than with those of pure touch.

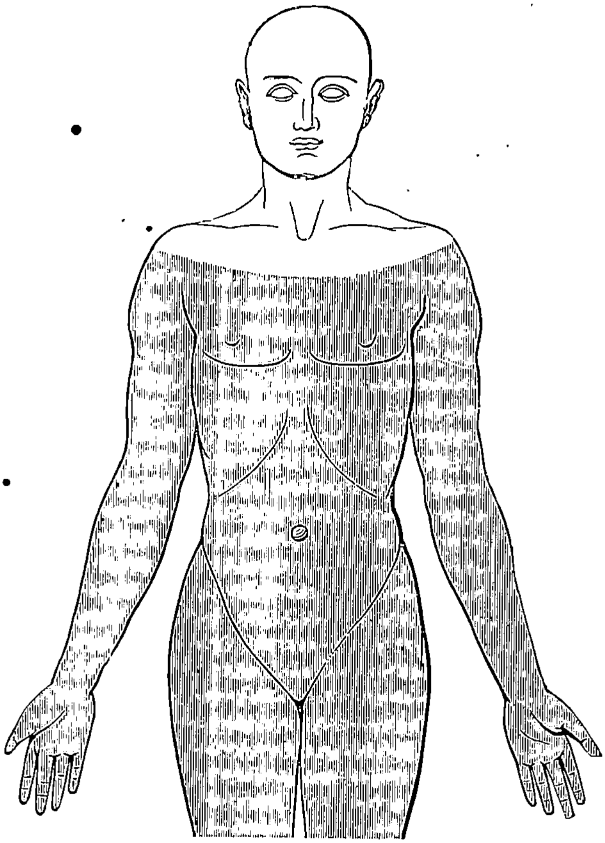

Fig. 1.

Turning next to the lower roots of the brachial plexus, we have cases such as those of which the anæsthesia is illustrated in fig. 4 (the dotted area was slightly hyperalgesic): in the patient from whom this drawing was taken the motor paralysis gradually extended up the cervical cord; on admission the intrinsic hand muscles and the flexors of the fingers and wrist were alone completely paralysed; on the second day the extensors of the wrist, and about the fourth day the pectoralis major, latissimus dorsi, teres major, and pronators succumbed. On admission "anæsthesia was nowhere complete, but was almost so below the level of the third rib, and in the upper limbs internally to a plane running down the centre of the arms and forearms to the styloid processes of the ulnæ, both before and behind." The progress of the paralysis was followed by a gradual

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[1] For the sake of brevity the *post-mortem* examination of these cases, most of which have been elsewhere published, are here omitted. In all the cases quoted they accord with and explain the clinical condition.

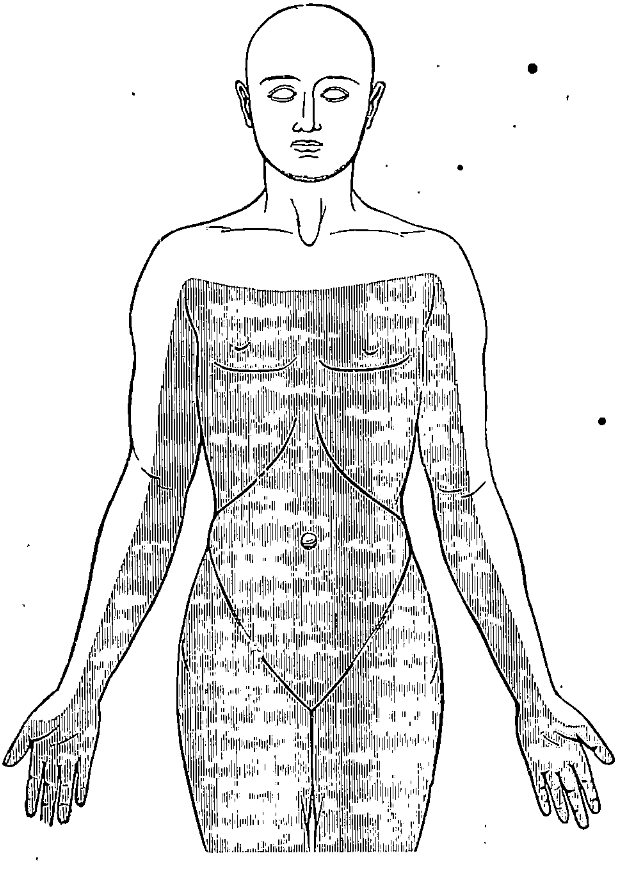

FIG. 2.

increase in the intensity and extent of the anæsthesia, until ultimately the whole of both upper limbs were involved. (Case 10 in "Surgery of the Spinal Cord.") In this case the boundary of the anæsthetic area was never well defined, but in another patient (*loc. cit.*, Case 12), from whom is taken fig. 5, "in the upper limb anæsthesia was limited with the greatest accuracy to the inner side of the arm and forearm, being bounded in front by a line running vertically from the front of the deltoid opposite the second rib, down the centre of the biceps and forearm to the middle of the wrist; similarly behind the boundary was a vertical line down the

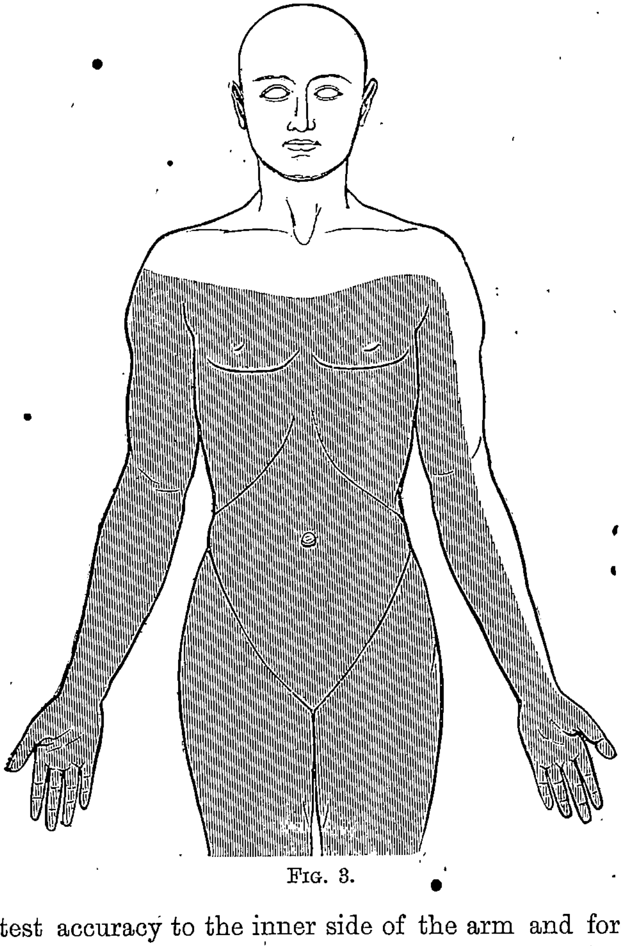

Fig. 3.

centre of the triceps and back of the forearm to the centre of the wrist. In the hand the limiting line sloped, both in front and behind, from the centre of the wrist to the cleft between the fourth and fifth digits, the little finger being completely anæsthetic, the others not at all so." Associated with this anæsthesia was total paralysis of the intrinsic muscles of the hand, the interossei, and extensor communis digitorum, and great weakness of the flexors and extensors of the wrist, especially of the former. The lesion was a "fracture-dislocation" at the junction of the last cervical and first dorsal vertebræ, and had therefore probably implicated the eighth cervical, first dorsal and subjacent nerve roots.

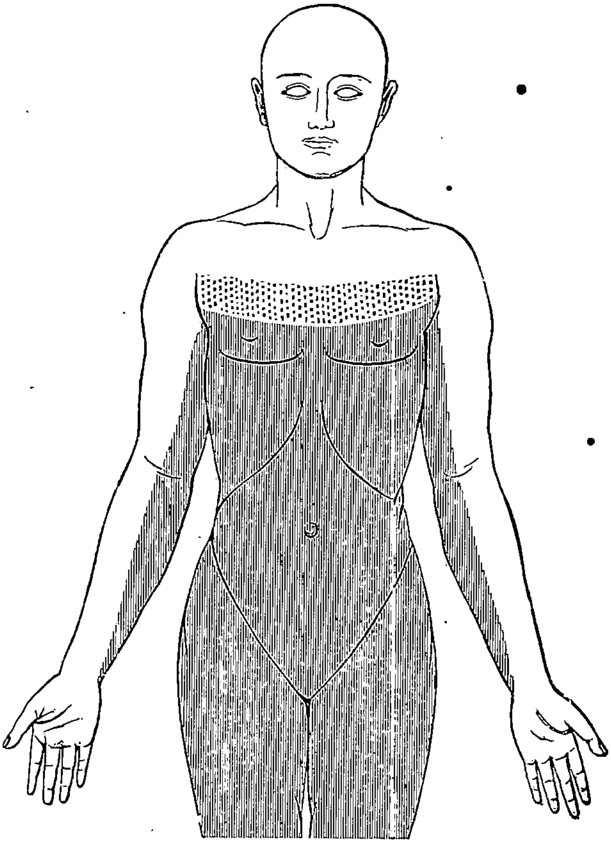

FIG. 4.

From the above it would appear to me probable that the sensory distribution of the eighth cervical and first dorsal is represented by the anæsthetic area in fig. 5 and that of the first dorsal in fig. 4. Head (p. 44) has quoted the case depicted in fig. 5 as an example of anæsthesia resulting from injury to the first dorsal roots only, but a comparison with the previous cases leads me to the belief that the eighth cervical is here also involved. The above cases must suffice to illustrate the proposition with which the discussion of the brachial plexus was commenced: others have presented themselves to me since these were published, but they have not led me to modify the conclusions.

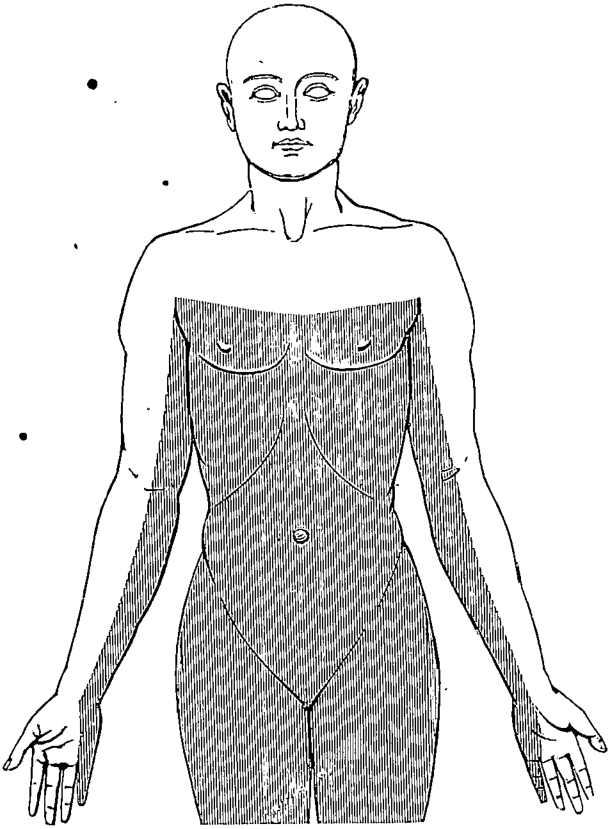

FIG. 5.

Turning now to the recent literature of the same subject, we find comparatively few observations, but such as have appeared confirm these views. Thus Starr (*Am. Journ. Med. Sci.*, May, 1888) gives the following in his table of the sensory distribution of the cervical nerves :—

5th. Back of shoulder and arm, outer side of arm and forearm to the wrist.

6th. Outer side and front of forearm, back of hand, radial distribution.

7th. Radial distribution in the hand ; median distribution in the palm, thumb, index, and one-half middle finger.

8th. Ulnar area of hand, back and palm, inner side of forearm.

1st dorsal. Inner side of forearm and arm to axilla. Some of these descriptions are difficult to follow without diagrams, but the coincidence of the two last with my diagrams is most striking. Bruns (*Deutsch. Med. Wochenschrift,* 1889) records two cases which bear out generally the above views. Mills (*Therapeutic Gazette*, 1889) gives Starr's table with modifications based on his own and other recent results, but in no way modifies the above quoted passages. Herter (*Journ. of Nerv. and Ment. Dis.*, 1891) records some interesting cases, and especially :—(1) Dislocation of sixth cervical vertebra ; softening of cord at level of eighth and to a less extent seventh cervical segment, " right arm, flexors and extensors of fingers and interossei completely paralysed, flexors of wrist very paretic ; left arm, flexors and extensors of fingers and interossei very weak, flexors of wrist slightly paretic ;" anæsthesia bounded by a line running down centre of arm and forearm, involving all the fingers and sparing the thumb and thenar region. (2) Crush of cord with serious injury to eighth segment, gradually returning to normal structure in sixth ; paralysis of intrinsic muscles of hands ; anæsthesia similar to last in arms and forearm but involving only the two inner fingers and the tips of the index and middle fingers. In both of these cases the anæsthesia is rather more extensive than in my cases with a similar amount of motor paralysis. Charcot records (*Archives* *de Neurologie*, 1891) a case of injury to the first dorsal root on the right side, in which the anæsthesia was beautifully defined in the areas shaded in fig. 6, the immunity of the second dorsal distribution on the inner side of the first dorsal being also here shown; there was considerable atrophy of the hypothenar muscles and interossei, to a less extent of the thenar muscles, and some wasting of the forearm; paresis of the muscles of the hand and forearm, except the extensors; small pupil and small palpebral fissure. This case entirely confirms the views above expressed as to the first dorsal distribution.

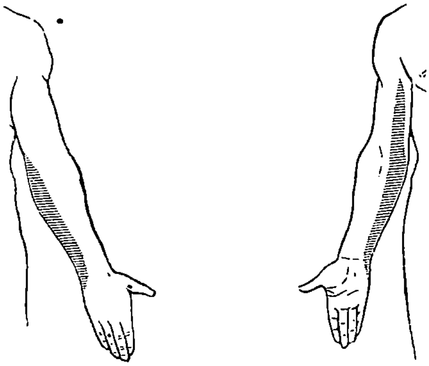

Fig. 6.

From Charcot, *Arch. de Neurologie*, 1891.

On comparing these areas with those obtained by Head's method, we find his first dorsal area to correspond very closely with that shown in figs. 4 and 6 of this paper, but Head's area is brought a little further on to the hand. This is the more remarkable as Head's areas tend generally to be smaller than the anæsthetic areas, and I cannot but suspect that his first dorsal region corresponds to at least a part of that supplied by the eighth cervical. And further, if we trace Head's areas downwards we find other evidences of a tendency to describe them as being a segment or part of a segment too high. Thus we find that he places the nipple in the area for the fifth dorsal root; whereas Sherrington states very definitely that, as a result of his researches, the nipple (in the monkey) "lies in the middle of the fourth thoracic field," which corresponds to the fourth dorsal of man; and "Quain" has it that in man the anterior cutaneous branches "from the third and fourth nerves are distributed to the mammary gland."

If, however, we turn to the lower part of Head's diagram, there appears to be a tendency in the opposite direction, that is, the areas are depicted too low. Thus it will be seen immediately that his first lumbar region corresponds more closely to that which Starr and others besides myself assign to the second lumbar, and anatomical dissection shows that the first lumbar nerve is mainly distributed above the line of Poupart's ligament, where Head has the twelfth dorsal, and that only a portion of its area lies below the ligament on the front and inner side of the thigh. Again, anatomy does not indicate that the twelfth dorsal descends so far below the ligament as Head has it. And finally the umbilicus probably lies, not as Head places it, at the junction of the ninth and tenth dorsal fields, but certainly no higher than the lowest part of the tenth. Thus Rüdinger (*Anat. der mensch. Rückenmarks-Nerven*) shows the eleventh dorsal opposite to the navel; "Quain" (9th edition) places the umbilicus between the tenth and eleventh; and Schwalbe has it opposite the tenth.[1]

For these reasons I am inclined to believe that Head has carried his lower roots too low down, and his higher roots possibly too high up, and that he has therefore represented too many segments somewhere between the fourth and the ninth dorsal fields.

These discrepancies are rendered the more interesting by the fact that, when he comes to the lower lumbar and sacral roots, where his enumeration starts from a fresh point, his areas agree almost exactly with my own.

We may turn now to the *lumbo-sacral* nerves, but here space will not allow me to trace out the cases by which I have arrived at what I take to be the distribution of the individual roots. Most of these were lesions of the cauda equina and were highly complicated, but they are somewhat fully discussed in BRAIN for January, 1888, in the *Medical Chronicle* for 1889, and in my "Surgery of the Spinal Cord." On the basis of these cases I drew out a series of illustrations of the probable cutaneous distribution of each root, and these illustrations were collated by me in the Jacksonian

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[1] For these anatomical references I am indebted to my friend Prof. Paterson, whose own authoritative opinion is that the umbilicus generally lies between the tenth and eleventh dorsal nerves.

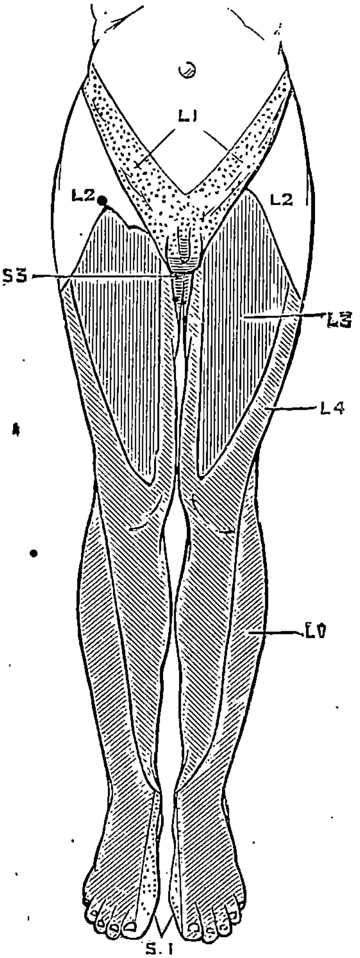

FIG. 7.

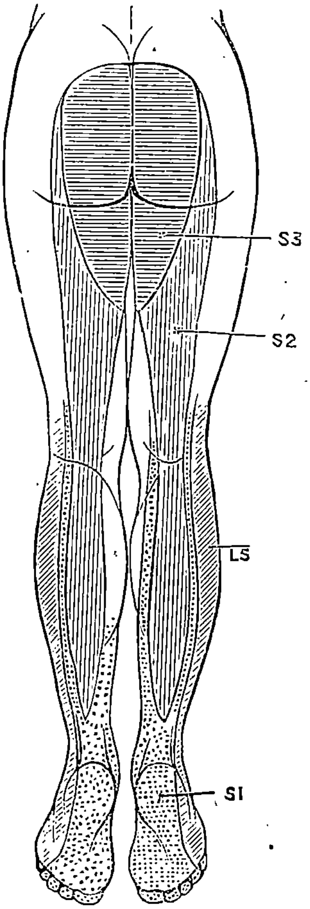

FIG. 8.

Fig. 7.—" L1, first lumbar ; L2, second lumbar, &c. ; S1, first sacral, &c."
Fig. 8.—" L4, fourth lumbar, &c.; S1, first sacral, &c."

essay, although only verbally described in the published work referred to. They are now combined in a single diagram (figs. 7 and 8), where an attempt is made to illustrate the whole of the evidence derivable from cases of spinal injury. It must, however, be fully understood that the boundaries of the areas represented in these figures are not to be taken as absolutely defined; they are merely general delineations of the usual distribution of each root. Thus in the case of the sole of the foot it is not attempted to depict the true boundary between the first sacral and the fifth lumbar, there being no evidence upon this point among my own cases; and in certain cases where there were two conflicting results or series of results the two sides of the body are differently shown, as in the case of the second lumbar, which has at times a well-marked distribution on the front and inner side of the thigh, whereas in other cases it takes little part in the supply of this region. Lastly it must be added that the fourth sacral and subjacent roots are not indicated, being placed too low down in the perineum for representation in these sketches.

From the time of the publication referred to there date numerous records, all of which more or less closely confirm the results arrived at. Starr (*Am. Journ. Med. Sci.*, 1888) briefly records the results thus obtained without comment, and a year later Mills (*Therapeutic Gazette*) again endorsed them. The latter also publishes several cases of lesions of the cauda equina, which confirm the statements made above (*Medical News*, March, 1890, and June, 1889). Church and Eisendrath (*Am. Journ. Med. Sci.*, 1892) record a case which is peculiarly interesting, as there was a crushing lesion of the sacrum, cutting off some of the roots of the cauda equina whilst it spared others: such a case is of special value, as most of our evidence is derived only from general pressure lesions of the cauda, and we therefore reproduce the diagrams given by these authors (figs. 9 and 10). They state that the anæsthetic area embraced "exactly and only the cutaneous distribution of the four lower sacral nerves," but it will be found to involve at least a part of the territory assigned by both Head and myself to the first sacral; at this we are not surprised on reading that uon operating for the relief of pressure "at the lower level of the first sacral vertebra the spinal canal was found, and the crushed-off termination of the dural sheath and cauda equina." As the first sacral nerves emerge below the first sacral vertebra we should expect them to be injured in a lesion at its lower level, and we would suggest that Church and Eisendrath's case gives a picture of anæsthesia involving the first and subjacent sacral nerves.

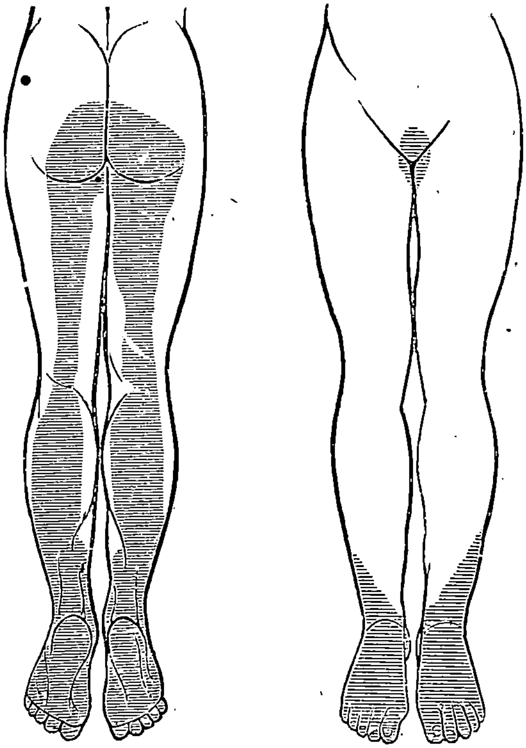
FIG. 9. FIG. 10.

Figs. 9 & 10.—From Church and Eisendrath, *Am. Journ. Med. Sci.*, 1892.

Lastly Starr (*Am. Journ. Med. Sci.*, 1892) has recorded six original cases and collected others, in which localised anæsthesia of the lower limbs was produced by lesions of the cauda equina or conus terminalis. He gives a large number of diagrams, which illustrate these cases in graphic form and, as a final analysis, he works out the distribution of several of the roots in the following figure, copied from his paper (figs. 11 and 12). This diagram differs very

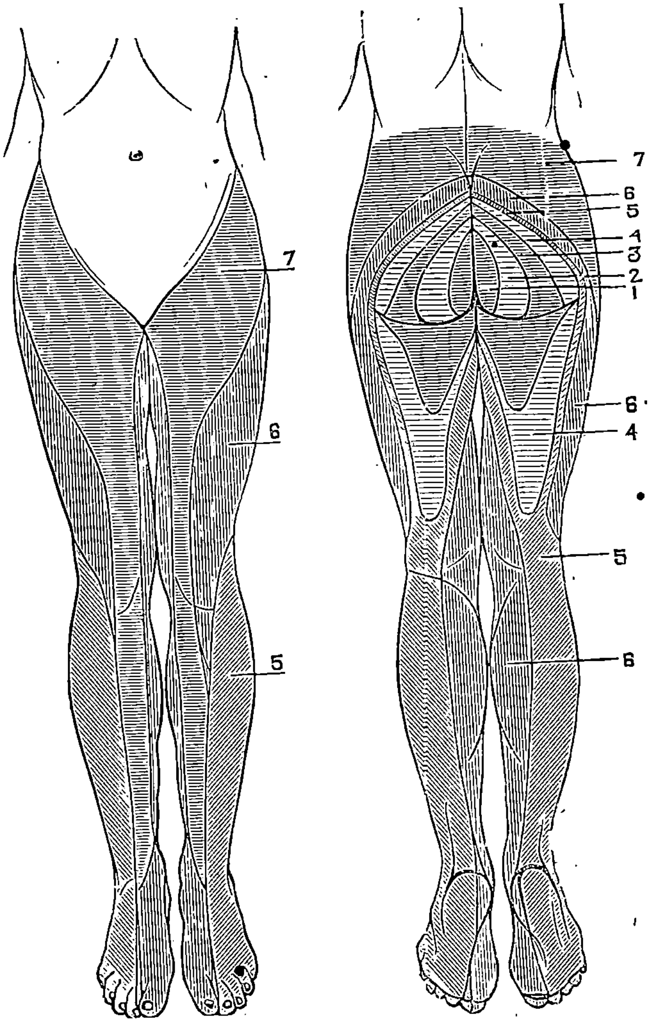

FIG. 11. FIG. 12

1. Sacral V. 2. Sacral IV. 3. Sacral III. 4. Sacral I. 5. Lumbar V 6. Lumbar III. 7. Lumbar II.

Fig. 11.—From Starr, *Am. Journ. Med. Sci.*, 1892.

materially from both Head's and my own, but it is obviously not by any means perfectly correct, as the entire lower limb is furnished with root areas, whereas the fourth lumbar and second sacral nerves do not appear at all, and we can hardly doubt that these roots have sensory branches.

In conclusion, there is one more point to which reference must be made. It will be noticed that in my diagrams and descriptions, I have throughout indicated that the cutaneous area supplied by each root always extends upwards towards the trunk, tending to reaching to both mid-venter and mid-dorsum. The areas thus appear in the lower limb as a series of roughly-shaped V's and Y's, each being wedged as it were into that which lies next on its distal side. Thus fig. 7 shows the third lumbar splitting the fourth, and the latter again splitting the fifth; whereas in fig. 8 we have the third sacral splitting the second, and the latter dividing the first; this arrangement is less obvious in the case of the remaining roots, but the tendency to it will be observed in all. Similarly in the upper limb it was assumed that the sixth, seventh, and eighth cervical provided long narrow strips of sensation from the trunk to the hand, where they spread out to occupy a wider area.

The reasons for this view were partly theoretical, partly actual observation. The primary theory was that so fully explained by Ross, who regarded the limb as a bud thrust out laterally and carrying with it layers of cutaneous nerve supply derived from each of the segments to which it was related. Then again, the fact that the fifth cervical and first dorsal nerves supplied long narrow strips of skin appeared to render it probable that equally long and possibly narrower strips were provided by the intermediate roots. How far the influence of these views has biassed my observation of facts it is difficult to estimate, but I still incline to the opinion that, in the lower limb at least, we can identify long strips of cutaneous supply given by each root. Thus in the cases recorded by Kahler, Eulenberg, and Starr and Lloyd there is a very narrow band of anæsthesia down the back of the lower limb, a band which I take to represent mainly the distribution of the second sacral nerve. This band, which unites the "saddle-shaped area," supplied by the third sacral, with the sole of the foot, supplied (according to Head and myself) by the first, has been several times observed by me, although it has always been in the form of a partial and not of a complete loss of sensation. Now I feel satisfied that this band is wider and its anæsthesia more marked if the anæsthesia extends to the first sacral or fifth lumbar nerves, and I have seen it become narrower and its intensity diminish when, during gradual recovery, these lower roots were regaining their function. This was well marked in Case 45 of my "Surgery of the Spinal Cord," although not clearly brought out in the diagrams—a remark which applies also to Case 32. The interpretation of this observation I have taken to be that the first sacral and fifth lumbar areas run down the whole length of the thigh on either side of the second sacral band.

Sherrington, on the other hand, in his experiments on the monkey, describes inter-digitating V-shaped areas similar to those in my diagrams, but denies the connection of the more distal roots with the mid-venter and mid-dorsum, and describes these segments as being "dislocated" by a lateral deflection into the limb of the mid-dorsal and mid-ventral lines.

It is perhaps too early to decide whether this view is entirely a correct one, but it must not be ignored that Sherrington bases his conclusions mainly on the fact that the hypothesis of an upward prolongation of every distal root area would imply a very great "overlap" in the limb. Although, however, the overlap would certainly cover many roots, it would involve only a very small cutaneous area, and it has yet to be proved that overlap does not depend at least as much on actual distance as on the number of root-areas covered.

ON THE EXACT SENSORY DEFECTS PRODUCED BY A LOCALISED LESION OF THE SPINAL CORD.

BY W. HALE WHITE, M.D.

THE following case seems to me worthy of record, for (1) it helps us to learn over what areas of skin sensation is perverted when there is a localised lesion of the spinal cord, and it is thus of importance in connection with Dr. Head's researches; (2) it illustrates what forms of sensation are the first to disappear when the cord is compressed, and it is therefore an aid to the elucidation of the paths of sensory impulses in the cord.

(1) The cord was compressed opposite the seventh dorsal vertebra, that is to say, at the point of origin of the eighth dorsal nerve. We ought, therefore, according to Dr. Head, to have found that the patient had loss of sensation below the upper margin of her eighth dorsal (middle epigastric) area. Reference to the figure will show that this was so on the right side. On the left there was loss of sensation below the upper limit of her ninth dorsal or supra-umbilical area. This difference on the two sides is probably to be explained by the fact of the cord not being compressed at exactly the same level on the two sides. There can, I think, be no doubt that this case strongly supports Dr. Head.

(2) We may from this patient perhaps learn something about the paths of the upward conduction of sensory impressions. The whole cord was compressed opposite the seventh cervical vertebra, but probably the lateral parts were much more compressed than the posterior, for on the sides of the cord there was some thickened inflammatory material which did not extend further back than the posterior roots.

In the first place, it will be noticed that over the area of skin corresponding to the whole of the cord below the point of compression, pain was interpreted as touch. This fact has often been observed before in similar cases, but nevertheless it is frequently overlooked, and cases are reported as suffering from cutaneous anæsthesia, when in reality they are only suffering from analgesia. Many authors assume that impressions of pain are different from those of touch, and that they ascend in different parts of the cord, founding this belief chiefly on cases such as the one here recorded. According to them, painful impressions are transmitted up in the lateral part of the cord, and simple sensations of touch in the posterior part. It is probable, however,[1] that "pain is no especial quality of sensation, but merely a special designation for the painful or unpleasant feeling that accompanies a very intense dermal sensation." On the first view we must assume that the pressure opposite the seventh dorsal vertebra so compressed the cord that it destroyed the functional activity of those fibres in it which transmit painful impressions; or on the second that those fibres in the cord which conduct sensations of touch were so compressed that their functional activity was lowered. This view is certainly supported by the fact that tactile sensibility was impaired—although it is true that the slightest touch was recognised—for the patient could not distinguish silk from velvet by the touch when they were applied to the feet, although a healthy person was able to do so, and with our patient it was necessary to remove the points of a pair of compasses before she recognised them as two, very much further apart than was necessary when the points of the same compasses were applied to the same parts of the skin of a healthy woman. But in favour of the first view is the fact that there is very strong reason for supposing that the lateral portions of the cord were more compressed than the posterior, and previously recorded cases of localised damage to the spinal cord appear to indicate that painful impressions are conducted up in the lateral columns, and those of touch in the posterior. I very much doubt, however, whether in some of the previously recorded cases the statement that touch was unimpaired is as valuable as it seems, for observers have often been content with saying that touch was normal when the patient could feel the slightest touch. But our patient could do this, nevertheless it was shown by the silk and velvet, and compasses, that her touch certainly was not normal.

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[1] Ziehen. "Physiological Psychology." Eng. trans., p. 133.

Over the whole of the analgesic area the patient could not distinguish between moderately hot and moderately cold objects applied to the skin, although she could between very hot and very cold. It has been observed in other cases of damage to the spinal cord, that when the capability of perceiving painful impressions, as such, is abolished, there is impairment or absolute abolition of temperature sense, and from them it has been inferred that sensations of pain and those of temperature are conducted in closely contiguous parts of the cord, and that as pain is probably transmitted upwards by the lateral columns, so probably are sensations of temperature.

It is especially interesting that both forms of muscle sense were unimpaired in this patient. She could tell an addition of 1 oz. in 12 oz. suspended from the ankle, and when I myself was tested I could do no better. Also she knew in what position her limbs were placed when she was blindfolded and I put them in any particular posture. These facts to some extent harmonise with the belief that the impressions of muscular sense and common sensibility ascend together in the posterior part of the cord, for this patient could feel the slightest touch, although in some particulars the sense of touch was impaired, and therefore on this view we must suppose that this part of the cord to some extent felt the effects of the compression.

The assignment of the conduction of pain and temperature to the lateral portions of the cord, and of muscle sense and touch to the posterior part, while no doubt it derives support from cases in which the cord was compressed from without, is difficult to understand when we study syringomyelia in which the pressure is exerted from the centre of the cord towards the periphery, for in them also the power of appreciating sensations of pain and temperature is lost before the power of appreciating those of touch and those of muscle sense. But syringomyelia is often more marked at the posterior part of the cord than the lateral, so that we should have expected that sometimes at least the ability to distinguish tactile and muscular impressions should be lost before the ability to tell those of pain and temperature.

The last point to which I would direct attention is that there was no delay in perceiving sensations of touch or temperature, although both these were impaired, which seems to me very difficult of explanation, for we should have expected that if some fibres were so damaged that they could not conduct impressions accurately, neither would they be able to conduct at the normal rate.

On the whole, therefore, although at first sight this case supports the teaching that sensations of pain and temperature ascend in the lateral columns, and those of touch and muscle sense in the posterior, on looking more deeply we see some phenomena of it which are not explained on this hypothesis, viz.—the inability to tell silk from velvet, the wide separation of the compasses necessary before the points were distinguished as two, and absence of delay in the conduction of tactile and normal sensations. Facts like these should make us pause before assigning definite tracts to sensory impressions.

Sarah Pretlove, aged 54, admitted into Miriam Ward, Jan. 12, 1893. Clinical assistant, Mr. A. H. Leete, B.Sc. The family and previous history are unimportant.

Present illness.—This began three months ago with a pain which encircled the trunk on each side from a spot between the umbilicus and the ensiform cartilage. There was no vomiting, and the pain was not increased by food. About a month ago the patient noticed a few slight jerks in the leg, and that she staggered when she tried to walk. This increased till the present time when the patient drags the legs so that she cannot walk. She says that the legs always feel cold. Within the past three months a swelling has developed in the mid dorsal region of the spine.

Condition on admission.—The patient looks feeble and older than she really is.

Motor Symptoms.—There is no paralysis of the lower extremities, and the power of flexing the knee joints is good, and the thighs can be flexed on the abdomen, and the feet can be bent, but both lower extremities are in a typical spastic condition, and show the clasp-knife rigidity when extended at the knees. It is the spasticity which prevents the patient from walking.

Sensory Symptoms.—(1) Tactile Sensibility. There is no delay in perceiving touch, nor any impairment of the power of localisation anywhere. But there is some impairment of tactile sensibility, for the patient cannot by means of the soles of the feet distinguish silk from velvet, although it was found that a healthy person could. Further, the sense of touch (as tested by the minimum distance at which the points of contact of the points of a pair of compasses are perceived is two) is impaired thus :—

On soles of feet minimum distance ... ... = 1 in.
,, right leg just below knee ... ... = $1\frac{7}{8}$ in.
,, left leg just below knee ... ... = $1\frac{7}{8}$ in.
,, abdomen ... ... = over 3 in.

(2) Common Sensibility. There is no delay in sensation, but there is impairment, for if the patient be pricked with a pin she interprets the experiment as a mere touch. This analgesic area extends from below a line following the downward slope of the 9th left intercostal and the 8th right intercostal nerves down to the commencement of the lower extremities on both the back and front of the trunk (see fig.) The upper limit is absolutely definite on the front and sides. On the back it is slightly indistinct. It extends over the whole of the lower extremities except a few small areas where the prick of a pin gives rise to a sensation of pain; there is one such area on the outer aspect of the middle third of the right thigh, and also one in a similar position on the left thigh.

No definite hyperalgesic area can be demonstrated, but it is probably not entirely absent. Attempts to locate it definitely lead to no constant result, but the results always lay between the 6th and 9th nerves. Pressure on the 6th right and 7th left intercostal nerves always caused pain. There is a very definite and constant girdle pain encircling the body. When asked to localise it the patient always places her hand over the epigastric and hypochondriac regions. The pain she describes is always present, and is of a dull heavy character.

(3) Temperature. There is no delay in perception, but over the whole of the analgesic area there is impairment of power to distinguish between heat and cold, but the power is nowhere

absolutely lost, nor is it ever perverted. On the chest and arms above the analgesic area there is no impairment.

(4) Muscle Sense. The patient when blindfolded can always tell the position her lower limbs are in when at rest, and also the position in which an observer may place them. When a broad bandage is placed round the ankle and a weight suspended from it, the least increase the patient could detect on either side was 1 oz. added to 12 oz.

Reflexes. The knee jerks are increased, the plantar reflexes are lively, the epigastric reflex is absent. Ankle clonus is present.

Micturition and defæcation are normal. There are no trophic changes.

Spinal Column. There is a considerable backward projection of the 7th to 10th dorsal spines, which form a prominent convex swelling.

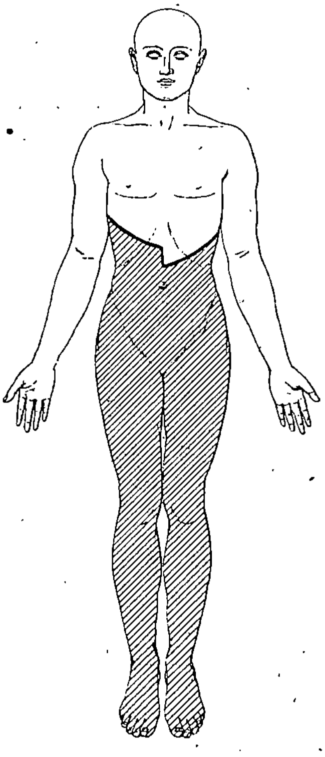
The respiratory, circulatory, alimentary, urinary systems are normal.

January 20.—Mr. Lane cut down upon the swelling in the back. The laminæ of part of the 6th, of the 7th, and part of the 8th vertebræ were removed. The cord did not pulsate. A spoon was introduced between the bone and dura mater, and pus welled up on either side of the cord, and on thrusting a probe between the 6th and 7th dorsal vertebræ much more pus welled up and some pieces of carious bone came away. A channel was then scraped between the two vertebræ, and an abscess cavity on the front of the spine was detected. This was thoroughly syringed out, and much pus flowed away. The cord commenced pulsating when the pus was let out. Drainage tubes were put in, the edges of the wound were brought together, and it was dressed with cyanide gauze.

January 23.—The patient, who has never thoroughly rallied from the operation, gradually sank and died at 10 p.m.

January 24. *Autopsy*.—In the chest on each side of the vertebræ was an abscess cavity; on the right side it was in contact with the 6th, 7th and 8th vertebræ, on the left with the 7th and 8th. These abscess cavities extended laterally from the vertebræ for about two inches under the pleura. The 5th to the 8th vertebræ were removed for examination. The body of the 7th vertebra was almost completely destroyed, the only living part of it being a small fragment attached to the 8th vertebra. The posterior third of the intervertebral disc between the 7th and 8th vertebræ was destroyed. The disc between the 7th and 8th was intact. There was no pus within the dura mater. The posterior part of the cord appeared quite normal. On the lateral aspects of the cord there was some thickened inflammatory material which extended to the posterior margins of the posterior roots, but, as just mentioned, left the posterior part of the cord free. Anteriorly it had extended over the cord, but to a less extent than laterally. Opposite the 7th vertebra the cord was soft and compressed.

There was healed phthisis at the apex of each lung, and there were scars of old gastic ulcers in the stomach.



DESCRIPTION OF FIGURE.

Over the shaded area and over a corresponding area at the back there is:—

(1) Analgesia. Pain being interpreted as touch.
(2) Impairment of tactile sensibility: (*a*) velvet not distinguished from silk; (*b*) points of compasses far apart called one.
(3) Impairment of power to tell hot and cold apart.
(4) No delay in perception of pain, touch or temperature.
(5) No inability to tell passive position of limbs.
(6) No inability to tell added weights.

ANGIO-NEUROTIC ŒDEMA.

(*With a record of five cases.*)

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THE occurrence of various transient vascular phenomena of the nature of flushings, erythema, urticaria, acne rosacea, &c., in dyspeptic and hysterical patients, as well as in girls at the menstrual periods and women at the climacteric, has long been recognized. The appearance of "Œdema in a limb, the subject of hysterical paralysis, and its disappearance with the recovery of power have, since the publication of Charcot's lectures, been admitted to be of neurotic origin." Bauke last year drew attention to the occurrence of circumscribed œdema of sudden onset and rapid disappearance in individuals of neuropathic tendencies without other signs of disease, and gave details of two cases. We record here five cases, in all of which the patients were free from any organic disease which would have had any influence at all on the production of this condition. In no case was the temperature raised, the œdema was painless in character and did not disturb the general health. An œdema of probably purely nervous origin is comparatively rare, and we have therefore thought these cases worth recording, and have offered some remarks on their general etiology and pathology, and have indicated the position they seem to us to occupy among the neuroses.

Case 1.—Peter M., aged 22 years, was under the care of one of us in the wards of Highgate Infirmary for compression paraplegia.

The patient was very neurotic, and from time to time had attacks of hysteria. He was probably a masturbator.

The following notes were taken during an attack of the transitory œdema we are now describing:—

April 19, 1891. A circumscribed erythema has appeared on the face around the right eye. There is a slight pitting on pressure, and a rise of local temperature perceptible to the hand. Temperature in rectum, 98.6.

Later in the day a redness with œdema was noticed over the left shin.

April 20. Erythema fading. Œdema gone.

April 21. There are swellings similar to those described above to be seen on both sides of the neck and on the back of the first phalanx of the right thumb.

April 22. The swelling has disappeared from the thumb, but the back of the hand and wrist is red and swollen. Towards evening the under lip became swollen, and at midnight there was a brawny red swelling on the back of the neck. None of the swellings are at all painful to the touch, and the patient was once or twice unaware of the swellings until they were pointed out to him.

The temperature taken every six hours was normal throughout the attack.

April 23. The patient is very hysterical, alternately laughing, singing, crying, and praying. He refused food for thirty-six hours. All trace of œdema and erythema has disappeared.

Early in May the patient's tongue became swollen. This swelling lasted eight or nine hours. He was then quite well for some months.

Case 2.—Ellen N., aged 14 years, a pale, nervous, and excitable child, was under observation in November, 1891, complaining of pains in the back and a feeling of general malaise. She drew attention to a red and œdematous swelling on her right elbow which had appeared that morning. Pressure on the swelling caused no pain. Patient stated that she had had a similar swelling over her forehead the previous evening, of which no traces remained. The patient was seen four days later and said that the swelling on her elbow had disappeared on the evening of the day on which she had first noticed it, and that it had been followed two days later by a swelling of the same nature on the back of the right hand, which also had disappeared in the course of a few hours. When seen again there was a distinct circumscribed œdema, with redness over the left ankle, hot to the touch, but not tender. (Temperature in mouth, 98.8.) One week later the catamenia appeared. After this no more local swellings were noticed, and the patient returned to her former good health. She was seen again six months later, and up to that time had kept quite well.

The following cases came under observation at the County Asylum, Rainhill, and we are indebted to Dr. Wiglesworth for his kind permission to publish them :—

Case 3.—James McG., aged 22, single, labourer, Irish, was admitted April 16th, 1892, suffering from adolescent insanity. His father, an excitable neurotic looking man, said that patient was the fifth of a family of eight and had always been "dogged and stubborn." Two of his sisters died of "decline."

On admission all his organs and urine were normal. He had widely dilated but reacting pupils. Ophthalmoscopic examination revealed no abnormalities. His palate narrow, somewhat pointed and high arched of "neurotic" type. Owing to his mental condition it was impossible to investigate satisfactorily his sensation and special senses. No anæsthesia was made out and no hysterogenic points ; he certainly stood pricking all over remarkably well, and seemed to take a positive pleasure in protruding his tongue and having it deeply pricked. He was quite aware of all that was done to him, and though pretending to be uninterested he furtively watched all movements made during his examination, very much after the fashion of a hysterical girl.

When left alone he would sit motionless with head bent and eyes fixed on the ground ; he would push away anything placed to intercept his gaze. He was generally neglectful of himself, dirty in his habits, and resistive.

He remained in this condition, with little change, for six months, when he was sent to the infirmary because he stood about in the cold with blue extremities and deficient circulation. Soon after this he became actively mischievous and dirty, and would sit about in contorted positions, such as "with his feet and legs held rigidly in front of him and his arms held out from his side horizontally," or "with the foot held by his two hands and thrust into his mouth," for half-an-hour or more.

October 24. His face was noticed to be swollen, somewhat red, but not inflamed, and there was œdema under the eyes and slight pitting on pressure over bony parts. There was no rise of temperature, which was taken every four hours, no albuminuria, no periostitis or dental trouble.

October 26. Swelling and œdema are little altered and have not extended beyond the face. There is no œdema or swelling in any other part of the body. Urine normal. Patient was kept on light diet for a few days, and the œdema gradually passed off, leaving slight pigmentation, but no other trace. The general health was unaffected. There was no erythema or urticaria.

December 18. Patient became very troublesome and mischievous, continually twisting his head round, biting his shoulders and arms; he would get out of bed at night and gnaw the linoleum on the floor. Swelling of his face with some reddening and œdema was again noticed, for which no cause could be found. His face felt hot, but there was no general rise of temperature and no œdema or eruption on the body. The œdema passed away again in a few days.

February 28, 1893. There have been no further attacks. He has changed little mentally, although a little more rational, and moving about when ordered, he is still fond of assuming *bizarre* positions, and conducts himself generally in a hysterical manner. We would draw special attention to this hysterical condition in both Cases 3 and 4, as having an important bearing on the causation of the vaso-motor trouble.

Case 4. George H., aged 22, single, tailor, was admitted May 22, 1892. A maternal aunt died in Rainhill asylum, of general paralysis. His brother said:—Patient was never a very bright lad but got on fairly well at school, which he left at 16, and worked at tailoring. Twelve months before admission he had an attack of influenza, which began with faints and severe pains in the head. He was laid up for a fortnight. Soon after this he became excited and talkative, and was continually preaching. After being three weeks in the workhouse infirmary for his mental condition he was still dull and stupid, and was sent to the country for a change, but did not improve, and has remained the same until now, except for occasional outbreaks of violence and excitement.

On admission there was no organic disease. Ophthalmic examination revealed no change in disc or fundus. His pupils were dilated, but reacted to light. His tendon reflexes were very exaggerated. He talks about religion and prays, but is incoherent, irrational and flighty, with a furtive smile and very self-conscious expression.

June 10. Patient is without doubt a masturbator, his mental condition is very variable, he is sometimes stuporose, at others restless and mischievous.

October 29. He was sent to the suicidal ward that he might be under continual supervision. He had threatened to throw himself out of the window; he was continually pulling at his finger nails and had torn two completely off. He had more than once tied his scarf tightly round his neck. He has picked some sore places on his head which are raw and granulating from continual irritation.

February 3. He has œdema and flushed face. There is swelling under the eyes and slight pitting on pressure over the frontal bone. On examination, his chest, abdomen, and the upper parts of the arms and legs are seen to be covered with a diffuse bright red punctiform rash resembling that of scarlet fever. The redness fades on pressure, but there is no œdema of any part of the body. Slight red lines arise after stroking the skin with the finger nail and remain visible for some minutes. No rise of temperature, no albuminuria, no increase of redness or swelling around the sores on the vertex.

February 5. The urticarial eruption has almost disappeared from the body and limbs, but an *artificial urticaria* can still be produced. The œdema of the face is less marked.

February 10. Œdema and urticaria completely disappeared.

February 20. Slight flushing and erythema of face. No œdema. A kind of *tache cérébrale* can still be obtained on the body.

His knee jerks are still very exaggerated and a patellar clonus can be most easily obtained, which is accompanied by a general tremor of the body almost approaching a rigor. With a little encouragement and suggestion no doubt a hysteroid fit might be induced.

February 28. No recurrence of œdema. He is in good general health and condition. We would add that influenza, appearing as an antecedent in this case, is becoming very frequent in the history of insane patients as an exciting cause.

Case 5.—Mary G., age 47, widow, charwoman. She has had three children, no miscarriages. Her husband died twenty years ago. She has been twice in Chester Asylum, for periods of three and four months. She was admitted December 2, 1892, suffering from dementia, secondary to climacteric insanity. Organs and urine normal; no skin trouble. Says she has not menstruated for six years. No signs of syphilis.

February 18. She has an erythematous swelling of the forehead and temples, extending slightly to the face, pitting on pressure, and feeling hot to the touch; there is also an erythema of the chest and abdomen, rather wider in distribution on the right side than the left. There is no hyperæsthesia or pain. No general rise of temperature. The urine was not examined. The erythema and œdema disappeared in a day or two, and the patient returned to her work in the laundry.

February 25. A slight patchy erythema without infiltration or œdema was noticed on the temples, and a patchy erythema of the neck and chest. On the abdomen in the hypogastric and neighbouring lumbar regions was found an œdematous and reddened condition of the skin, somewhat irregularly distributed, the margin of the affected portion being fairly sharply defined. The whole area affected was an irregular surface of about 12 by 10 inches. Temperature taken in rectum, 99.2. Patient complained of slight headache and a "feeling of water rushing up and down in the head."

February 26. Infiltration of skin of abdomen disappeared, leaving the skin slightly loose and wrinkled and a little pigmented. No erythema remaining in the abdomen. Slight traces of erythema remaining in the face. Urine, sp. gr. 1015, acid, a trace of albumen, no sugar, no casts, no lithic deposit. Temperature normal. Says she feels better in her head, but "the water still rushes up and down."

March 1. No remains of erythema or œdema. She feels quite well.

No friends have visited this patient, so that no history has been obtained, and the presence of heredity in this case is uncertain. The patient never heard of any of her friends ever having been in an asylum.

Etiology.—Numerous observers, for the most part continental, who have noticed this condition of fugitive œdema have implied their opinion of its origin in such terms as "nervous," "angio-neurotic," "neuropathic"; and we ourselves are convinced of the evidence of a strong neuropathic taint in all our cases. We therefore think that among the chief predisposing causes should be placed hysteria, hystero-epilepsy, neurasthenia, and the allied emotional states. Quincke, Strübing, Valentin and Osler think that heredity is an important etiological factor, and we have been able to obtain histories of a nervous disease although not of a similar affection in two of our cases.

First among the exciting causes, we would place psychical disturbances, and in this opinion we are confirmed by numerous authorities such as Bauke, Charcot, Küssner Bitot, E. Wagner, Napier, Richt, and Lesser.

Max Joseph, who records three cases of this affection, thinks that alcoholism plays a prominent part as an exciting cause. In his cases, all of whom were dipsomaniacs, abstinence from alcohol brought disappearance of the swellings, but when excesses were again committed the œdema reappeared. Other exciting causes we would instance are—(i.) The onset of puberty—Case 2 and Börner. (ii.) The climacteric—Case 5 and Börner. (iii.) Gastric irritation—Cases 3 and 4 and Strübing. (iv.) Exhaustive nervous drain as masturbation, &c., during the unstable time of adolescence—Cases 3 and 4.

Cutaneous distribution.—As will be seen by references to the clinical cases recorded, the skin-distribution of angio-neurotic œdema, like urticaria and unlike herpes zoster and some forms of psoriasis, is irregular.

Pathology.—That the vaso-motor system is greatly affected by emotional states is well known; the phenomena of blushing, flushing, and sudden pallor are so familiar to us all as to be looked upon as indications of certain normal mental conditions. But in any morbid change in the mental state such as is met with in hysteria and hystero-epilepsy, the influence which the vaso-motor system of nerves has normally over the calibre of the arteries is markedly increased. Examples, of course, are daily met with in the pale skin of an hysterical girl which does not bleed on pricking, and in the flushings and sweatings that are seen in women at the menopause. It is only to be expected then that as we ascend the scale in abnormal emotional states so we find stronger evidence of loss of control over, or greater irritability of, the vaso-motor system. It being admitted that neurotic hyperæmias exist, the question arises as to whether the dilatation can be carried a stage further, still under purely nervous influences, and a neurotic œdema be produced. We venture to think that this question may be answered in the affirmative if both clinical and experimental facts be taken into account.

Taking first into consideration what we believe to be clinical evidence in support of the view we advance.

Gowers has recorded a case of trigeminal neuralgia in which all the veins of the affected side of the face became distended during the paroxysm, and another case in which cranial neuralgia was accompanied by an œdematous swelling of the whole scalp. In cases of neuralgia Anstie has noticed a general arterial dilatation during the paroxysm, which he has demonstrated by sphygmographic tracings. Numerous cases of vaso-motor disturbances following diseases of the spinal cord are on record. Gowers states that they are chiefly seen in cases of disease of the intermediate grey matter and Leyden has noticed considerable œdemas develop very rapidly in cases of acute myelitis. Crocker mentions the case of a lady "in whom the advent of strangers produced an urticaria, and this sensitiveness increased until a knock or a ring at the front door would determine an outbreak." This case is of extreme interest as an example of the great irritability of the vaso-motor system in the emotional states, and we shall discuss later the near relationship that some forms of urticaria bear to angio-neurotic œdema.

From the experimental point of view also, evidence is not wanting of the existence of œdema caused by nervous changes. Ostroumoff produced œdema of the tongue of a dog by irritating the peripheral end of the divided lingual nerve by induction currents of gradually increasing strengths, and Gergeus observed that frogs whose spinal cords had been destroyed became œdematous under certain conditions.

But a still more important experiment is that of Ranvier who ligatured the vena-cava on one side and did not obtain venous stagnation in the limb until he had also cut the sciatic nerve on that side. It is fair to say that Sonitschewsky differs from Ranvier, and states that in cats and rabbits very slight œdema indeed results from this operation. But Ranvier's work has recently been repeated by Horsley and Boyce, and they obtained the same result as Ranvier in nine out of twelve dogs experimented upon.

Finally, Tigerstedt and Santesson have given practically conclusive evidence that the transudation of living fluids through living membranes is not a mere physical phenomenon solely due to changes in pressure.

We think, therefore, that we are justified in assuming that the case for an angio-neurotic œdema is fairly made out —that is to say, that in a certain class of cases an œdema occurs which is due to a direct influence of the nerves on the vessel-walls causing an increased permeability. And we also think that a distinction may be drawn between the œdema of neurotic origin, which is almost certainly due to an increase of nerve excitation, and the œdema such as was observed by Gergeus in paralysed frogs, which is very possibly due to a cutting off of the vaso-motor nerves from their centres, or in other words, to a loss of vascular innervation. The sequence of events in the production of this neurotic œdema is probably as follows:—A local paralysis of the vaso-constrictors or a reflex stimulation of the vaso-dilators, causes a dilatation of the vessels of the subcutaneous tissues, which is followed by retardation and stasis of the blood. An exudation, usually serous, sometimes sanguineous, then occurs, producing an œdema, but the œdema is not sufficiently acute or sufficiently localised to cause any lifting up of the eperdermis as a whole, and the resulting condition may be fitly termed an *abortive urticaria.*

Quincke, Dinkelacker, Rapin, Minich, and Osler have all noticed cases in which this condition of fugitive œdema was present, and in which there were subsequent attacks of genuine urticaria.

Strümpell also remarks on the near relationship that angio-neurotic œdema appears to bear to urticaria, and his view of the pathology of the former affection is that it is a transition stage between a vaso-motor and a vaso-trophic disturbance. In all the cases now recorded by us it will be observed that a local stimulation produced a marked vaso-motor change—sometimes merely a *tache cérébrale*—at others a factitious urticaria, and in one case a genuine urticarial eruption was met with in another part of the body.

It has been suggested that in urticaria the formation of the wheal is the result of the contraction of the muscles of the skin, and it is just possible that in angio-neurotic œdema no wheal is formed owing to the want of tone of these muscles. Considering the neurasthenic state of the majority of the patients in which angio-neurotic œdema occurs, such a loss of tone could easily be conceived.

Another point in common between angio-neurotic œdema and urticaria is transient albuminuria. Lente has noticed this in the latter affection, and we have observed it in Case 5.

Diagnosis.—This is, as a rule, a very easy matter, especially if its intimate connection with urticaria be borne in mind. Some clinicians, however, seem to have confounded this affection with the non-febrile form of erythema nodosum—for instance, Bitot and Simon have noted tenderness of the swelling, which is never present in neurotic œdema, and Küssner has described it as occurring with a rheumatic attack. On the other hand, Lewin, Auspitz and Schwimmer, have stated their opinion that the non-febrile form of erythema nodosum is dué to a vaso-motor disturbance.

In conclusion, we have endeavoured to shew that the symptom is one of the vaso-motor neuroses, and the importance of recognising it lies in the fact that it may be an additional guide in the diagnosis of neurotic cases, since it is an index of an unstable nervous system. Measures for its treatment should be directed towards the central nervous system after all sources of possible peripheral irritation have received attention.

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THE RELATIVE IMPORTANCE OF THE MINUTE HISTOLOGICAL FEATURES OF THE BRAIN CORTEX IN GENERAL PARALYSIS.

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THE perennial interest of general paralysis must be my excuse for a short essay on the minute pathology of a morbid condition which has engrossed the attention of the best observers in psychological medicine both here and in other lands. It is not my intention to go over and describe once more the morbid changes patent to the naked eye of any observer; nor even to give a detailed and complete account of the appearances which have been recognised under the microscope, and described with a fulness and accuracy not to be improved upon. The exact significance of the changes has been, and still is a matter of dispute. The relative importance of the pathological features of the different structures of the brain cortex still furnishes a field for investigation, and the determination of this point may lead to a fuller and more exact knowledge of the ætiology of the disease. This attained, an important advance may be looked for in the therapeutics of general paralysis, when recognised at an early stage.

It will be necessary for me in the first instance to sketch the appearances which I conceive to be typical in the cortex of the general paralytic, not so much for the sake of the description itself, but as affording the necessary peg on which to hang the subsequent remarks. Here I shall follow Bevan Lewis's description of the paracentral cortex. This locality is chosen because its arrangement into five layers is easily recognised in the normal brain, while the pathological changes are, on the whole, as distinct as in the frontal cortex.

Description.—To begin at the periphery, the pia mater is thickened and cellular and the epicerebral tissue is sclerosed and fibrous. The *first* layer of grey matter is thinned and contains numbers of enlarged Deiter's cells or "scavengers," which are frequently so numerous as to form a felted network with their processes. The walls of the vessels running through this layer are cellular, either through proliferation of the cell constituents of the wall or exudation of leucocytes into the perivascular space; but kinking of the vessels and cellularity are hardly so marked as in the deeper levels.

In the *second* layer of grey matter "scavenger" cells are present, though in diminishing numbers; the small polygonal nerve cells are better preserved, as a layer, than any others. Their protoplasm is granular and fatty, but the nuclei are distinct and well stained.

In the *third* layer the vessels begin to be very cellular and kinked or irregularly dilated. The ground substance is granular and cloudy, with little or no trace of nerve fibres. It has a blurred or ground-glass appearance. Scavenger cells are present especially in the neighbourhood of the vessels, though not as a rule in the numbers in which they infest the first layer. On cursory examination there may appear to be nothing but the turgid cellular vessels, scavenger cells with their processes, exuded leucocytes, and granular ground substance with a certain amount of *débris*. It is only after a more careful inspection that much of the latter is seen to be composed of remnants of smaller and larger pyramidal nerve cells. These have an irregularly spheroidal shape, and have totally, or almost entirely, lost their processes; they have a softened, half dissolved look, a very indefinite outline and a granular badly stained protoplasm. The nucleus is usually shrunken and insignificant in appearance, or else it may be swollen and distorted.

The *fourth* layer, consisting of large pyramid and ganglion cells, is greatly degenerated. It would appear that where general retrograde changes are taking place in the cortex, this layer is certain to present the most obvious and most advanced morbid characters. This is true, not only of general paralysis, but is seen in chronic mania, in dementia, and even according to my observation in epilepsy, although here I cannot cite Bevan Lewis's authority in my support.

In the *fifth* layer of spindle-shaped or irregularly triradiate cells of a size not far removed from those of the third layer, degeneration is pronounced. It is here that scavenger cells re-appear in force, and especially where the grey passes into the white matter. Very often where these peculiar cells are scanty elsewhere, they may be fairly plentiful in these situations and even in the parts of white matter most remote from the cortex.

In the normal brain, or one slightly altered by disease, the grey matter passes rather gradually into the white. One may see what may be described as "outpost" cells extending some little distance into the white matter as solitary nerve-cell units. They are small and difficult to define from the prominence of the surrounding endothelial nuclei. These cells are very apt to suffer and disappear in general paralysis. The degenerations of the nerve cells in the fifth layer are generally those of the other layers and equal in degree to those of the *third* layer.

The cellularity of vessels in the white matter and the presence of spider cells may be mentioned, but these changes are probably of secondary importance and need not detain us here.

To the naked eye the diminution in the thickness of the cortex is plain in general paralysis. Under the microscope the diminution can be explained by the dissolution of the nerve cells and the disappearance of their processes, which implies the destruction of a vast amount of the commissural conducting fibres of which the ground substance is largely composed. The described conditions render it evident that in many instances it is hard to say where one of the different cortical layers begins and where it ends, except in the case of the first and second. Here only the demarcation remains distinct. The foregoing description may be verified easily in a typical case, or what for convenience may be considered as a typical case on the authority of Bevan Lewis and others. The variations in the microscopical appearances of different cases are considerable, and may have helped to foster the idea that general paralysis is not a pathological or clinical entity. But the use of the term "typical" can be justified by the fact that typical cases are by no means uncommon, and that in every case, one or other of the principal histological changes will certainly be found. In most cases all will be present in a certain degree.

From a clinical point of view several kinds or varieties of general paralysis may be distinguished. In one case a maniacal onset or a well marked stage of exaltation is followed by a gradual enfeeblement of body and mind frequently accelerated by epileptoid seizures. Dissolution eventually occurs from exhaustion, caused by the latter, or from intercurrent disorder. Another may have exhibited no stage of exaltation, but a more or less rapidly advancing dementia, while in a third the mental phenomena may have been those of melancholia. One might expect from this to find at the necropsy some condition corresponding more or less constantly to the varieties and duration of the disease. As far as duration is concerned it is true that differences are found; where symptoms have been of long-standing, the brain is likely to show more general atrophy and greater accumulation of fluid both superficially and in the ventricles. But otherwise it is not found that degrees of congestion, or pachymeningitis, or adhesion of membranes have any special relation to mental symptoms, and it would be quite impossible to say what special features were displayed by the patient's disease from an examination of the cortex under the microscope.

The method of examining fresh brains microscopically which has been elaborated at the West Riding Asylum has been an enormous advantage in the study of the cortex in disease. By this method and staining with aniline blue-black are obtained results which for clearness and accuracy are not nearly approached by hardening methods. But the process presents considerable difficulties in practice. We not unfrequently find that complete sections of the cortex are hard to obtain; the peripheric portion has an unconquerable tendency to fray away, or perhaps after the sections have been cut with apparent success, on removal from the stain they are found to have taken it up excessively and coarsely, and have a blurred or dragged appearance. This is a frequent occurrence in general paralysis, and it becomes a matter of difficulty to decide how far the condition is actually morbid and how far due to faulty sectioning. The difficulty is indicated by the fact that one section may be very much better than another, and even one part of a section may be better than another. From a considerable experience in the preparation of brain sections, not only in general paralysis, but also in epilepsy, I should be inclined to say that the blame for the unsatisfactory appearances should be equally divided. That they are "artefacts" is shewn by the occurrence of one or two well preserved sections, among possibly a dozen bad or indifferent. But it is also evident that the preponderance of the latter is due to some peculiar change in consistence of the cortex which requires a very exact degree of freezing. If this is not attained, the faulty consistence results in a dragged appearance of both cells and ground substance, as I have mentioned, and renders appearances deceptive and description useless.

Another difficulty consists in the patchy nature of the pathological changes. The morbid processes are not uniform, nor do they shade off gradually into the normal, as one passes from one region of the brain to another. Not only in adjacent convolutions, but in different parts of the same convolution and the same section we may see the most marked cell degeneration, or vascular change, or scavenger cell proliferation, side by side with a condition comparatively little removed from the normal. This makes it impossible without cutting and examining an inordinate number of sections to say with certainty that some special pathological feature is absent from the brain *in toto*. The feature that occurs to me just now is the scavenger cell which is rightly considered when present in large numbers to furnish a highly characteristic appearance in general paralysis.

A few remarks on the nature and appearance of "scavenger" cells may not be out of place. In a normal brain the cellular elements of the connective tissue comprise here and there a corpuscle which can be distinctly seen to possess numerous fine processes—the Deiter's cells. These may be considered to furnish the normal type of the greatly enlarged and enormously active lymphatic element which from its numbers would appear to take origin, not only from cells which can be seen to possess processes, but also from those whose processes are invisible. The "scavenger" cell varies in appearance according to its stage of growth and the period of the disease. *A priori* reasoning would lead us to expect these products of a morbid activity to have distinct periods of growth, of full activity, and of decadence, and this is fully confirmed by microscopic examination of a number of cases. In describing a typical scavenger cell at the zenith of its power we should find it a protoplasmic cell of many processes. These latter vary much in length and degree of straightness, but their outlines are very distinct, and one or more of them is always attached to a vessel wall in the vicinity, though this may not appear in the section. At the point of junction can be seen a small collection of *débris* and pigment. The processes, which are doubtless hollow tubes, are more defined and take on a deeper stain with aniline blue-black than the body of the cell. The body of the cell is composed of a granular, sometimes slightly pigmented, mass of protoplasm. The shape is irregularly spheroidal and the nucleus is not always to be distinguished. This may be due to actual disappearance or merely to an excentric position. The size of the body of the cell varies considerably even at the period of greatest activity, but they may be so greatly developed as to surpass the mutilated remnants of the adjacent nerve cells, *e.g.*, the pyramid cells of the third layer. The general appearance gives the impression that the "scavenger" cells are attacking and breaking down, in fact actually battening upon the nerve elements. We shall return to discuss this point and its limitations later on.

In some cases on examining a good number of sections we may be unable to find a single adequate representative of the "scavenger" cell, though other pathological conditions may be fully developed. For instance, the disappearance of nerve cells and their processes may be quite as marked as I have described in the typical section, and the vessels may be kinked, aneurysmal, and cellular, and yet not a scavenger cell to be seen. This strangely negative appearance raises the question of the significance and nature of the scavenger cell, and of its action here and in other morbid conditions. The origin of these elements in the Deiter's cells is not likely to be contested, nor could they well be considered as anything else than a hypertrophy of the finest lymphatic element of the normal brain. Their appearance and connection plainly betray their character, and their presence in association with broken down cell elements suggested the descriptive name of "scavenger." As a descriptive name it is a good one, and the analogy can be pushed to the full. The function of a scavenger is to remove refuse, effete and useless material. But Bevan Lewis enlarges the function of these cells still further by attributing to them an active and aggressive part in the production of the diseased condition. He figures them as attacking and disintegrating the nerve cells with their processes which he regards as suckers, not in general paralysis alone, but also in alcoholic insanity. From what I have seen and from what I have said above concerning their not infrequent absence, it would appear to me that the rôle of these cells is probably a secondary one. The function of their originals is doubtless to remove the effete material produced by the ordinary metabolism of the nerve tissue. Is not the stimulus of a vastly increased catabolism sufficient to account for the overgrowth of this fine lymphatic element? The function of a scavenger is to remove rubbish, not to produce it. I concede the analogy established by their title to the full, but have been unable to go further and recognise an originating and aggressive action in these cells. If their aggressive and originating action were a reality we should hardly expect to find nerve cell degeneration without their presence. We should also expect to find the scavenger cell in the greatest prominence where the degeneration is best marked. But this is not the case. The most typical nerve cell dissolution may be seen without a single scavenger cell in evidence. In fact it is, as mentioned before, usual to find a much larger number of these cells in the superficial cortical layer where true nerve cells are wanting and at the junction of the grey and white matter than in the third and fourth layers where a more marked degradation has taken place in all the nervous elements.

It may be asked why this tendency to degeneration is exhibited in such a special degree by the cells of the third, and more particularly the fourth cortical layer. The explanation is a quasi-mechanical one. It stands to reason that the processes of nutrition in a cell are conducted with an ease or difficulty corresponding to the extent of surface exposed to the nutrient fluid as compared with the bulk of the cell contents. The smaller the cell, the larger relatively is the surface it possesses to subserve the processes of nutrition, and the more easily do the necessary anabolic and catabolic changes proceed.

Ceteris paribus, a converse statement may be made with reference to large cells. Hence, when there is a general inadequacy of, or strain on the nutrition of the nerve elements, it is the largest cells which break down the soonest. And it is possible that the size of these giant and large pyramid cells connotes a higher differentiation in development, and a corresponding diminution in reparative power. The degeneration of these elements is so common a feature in morbid brains that I fail to see any other explanation that is satisfactory. In correspondence with this view we often find in general paralysis that these large cells are much broken down and may be converted into *débris* and unrecognisable, while the smaller pyramidal cells of the third layer are easily to be discerned though, of course, much altered.

The removal of effete material is as essential to a healthy function as a proper supply of nutriment. In considering the want of relationship displayed between the degrees of nerve-cell dissolution and the presence of scavenger cells, can it be said that the latter are to blame for the degenerative processes in the nervous tissue? Is it not just as probable that this vast hypertrophy of the lymphatic element is the only method the brain has of keeping the nerve elements functionally active? The fact that this vast hypertrophy is an important sign of a diseased brain does not prove it to be essentially noxious. In chronic renal disease or general arterial sclerosis, hypertrophy of the left ventricle is a common accompaniment. This hypertrophy is undoubtedly a departure from the normal and a valuable sign of the presence of these diseases, but its object is in reality a beneficial one. It hypertrophies merely in order to enable the circulation to be carried on efficiently in the face of a generally increased resistance to the blood current. If untoward circumstances occur such as cerebral hæmorrhage it is the ruptured vessel which is to blame. Similarly, may we not say that the probabilities are large that scavenger cell formation is a compensatory process? It is only in cases of some standing that this formation is well displayed, while again in those that have survived long, the activity of the lymphatic element diminishes and a sclerosis replaces the previously well developed network of protoplasmic cells and processes. Why the occurrence of these cells should be so uncertain both in quantity and locality is difficult to say. The doctrine of individual idiosyncrasy is a convenient refuge. Similar agents acting in different organisms produce different results and act with varying severity. This will seem rather a commonplace explanation, but it is as good as I am prepared to offer. It would not be wise to re-classify general paralysis on the ground of certain histological differences in the brain, any more than it would be on the ground of variety in the clinical features. While one dies in epileptoid convulsions at an early stage, another succumbs to exhaustion after enduring three or four years of gradual decay. In spite of all, the disease is probably as much an entity as lobar pneumonia or enteric fever. It is a symptom-complex, to use a Germanism, in which one phase or constituent may predominate. Nor can the exciting cause be relied on to decide whether we have to deal with a case of true general paralysis or not. Alcoholic insanity is a form distinct from general paralysis, but there are cases in which a condition indistinguishable from general paralysis follows on this form of brain poisoning, just as ordinary maniacs or melancholics may develop a similar condition. A history of previous syphilis or excessive sexual indulgence are doubtless predisposing causes, though not essential antecedents. In some, an accident or injury to the head appears to have been the starting point. Mental worry in especial is allowed to have a great, if not pre-eminent, causative action. But if all these are recognised as ætiological factors, why should we deny that other irritants may actually produce the disease? If the irritation results in symptoms like those of general paralysis and similar microscopic appearances, can we not say that the disease has been actually produced though in an unusual way? A case of hydatids of the brain occurs to me, in which the irritation spreading from the base of the brain had produced a condition diagnosed as general paralysis during life, and very similar microscopic appearances in the cortex.

This digression has interrupted a consideration of the question I desire especially to bring under notice. What is the essential and primary lesion of general paralysis? There must be some changes in the nerve tissues of the brain cortex of the general paralytic even in the early stages, while the diagnosis remains in doubt, though the patient is plainly disordered in his mind. In what I have said above are embodied some of my reasons for not accepting the scavenger cell as the origo mali. The other two appearances of paramount importance are seen in the vessels and the nerve cells. These changes go, to a great extent, hand in hand; where the vessels are most thickened and cellular the dissolution of the surrounding nerve tissue will be most evident—as a rule. But this, like most other conditions, is by no means invariable. The nerve cells in a certain part may be fairly preserved with a marked proliferation of the cells of the vessel wall, and, *mutàtis mutandis*, the changes in the vessels in another case, or in another part of the same brain, may be quite insignificant while the cells and ground substance are seriously disorganised. In any case according to our present knowledge the intellectual phenomena and the motor disorder must be put down as due to failure or perversion of function in the proper nervous tissue itself. It matters not whether the abrogation is produced by something acting directly on the nerve tissue or indirectly through lesions of the apparatus that supplies its nourishment.

As Gowers[1] in a recent communication puts it, "Whatever be the original process" (*i.e.*, in nervous disease) "the symptoms themselves invariably depend on minute changes. If the primary morbid process is a massive one, as a tumour of the brain, the symptoms which manifest it presence and declare its character are of a minute nature." . . . . "The effects of a gross specific disease such as an artery causing softening may differ very little from those of a degenerative specific disease."

If the disease, then, of body and mind is accompanied by a degeneration of nerve cells which can always be recognised, whether the peculiar vascular and lymphatic conditions are in evidence or not, does it not justify the presumption that the essential and primary lesion is one of the nerve cells and their processes? It is quite possible that the same cause which produces the break-down of the nerve cells and their inter-communicating fibres causes simultaneously the cellular proliferation of the vessel walls. It may be that the vascular and lymphatic conditions when well shown are the most *characteristic* appearances in general paralysis. That does not necessarily imply that they are the essential and primary factors at fault. Scavenger cells are seen in other conditions; in alcoholic insanity, in senility, to a certain extent in epilepsy and in acute meningitis. The appearance of thickened, proliferated and cellular vessel walls is not so common a feature and as far as I know is not met with in a well marked state in any other condition of brain disease. But as this feature is not so constant as the dissolution of the nervous tissues to which ultimately the symptoms of the disease must be referred, I repeat that the probability is in favour of a purely nervous origin.

What the actual cause may be we are not in a position to say as yet. It may be that it is some organic poison which under certain conditions has the power of attacking and a special affinity for the higher nervous tissues. Not all that have led a fast life and "burnt the candle at both ends," or undergone great mental strain and worry, become general paralytics. It is no more than a small percentage that does so, although they may suffer in other ways. Though alcohol has an affinity for nervous structures, it is only a small proportion of the intemperate that undergo peripheral neuritis. The mousy smell in general paralytics, not only of the surface but of the whole body, would seem to lend some support to the theory of a quasi-septic intoxication being the originating evil. Besides, we have an analogy in influenza. In one or two cases which have come under my notice, with acute dementia as a sequela, the softening of the brain cells was remarkable, and appearances pointed to their rapid dissolution. The vessels did not present any notable feature beyond a light yellow staining, due in all probability to the solution of blood pigment. Scavenger cells were not present. The conclusions, then, may be shortly summed up as follows :

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[1] Address on Neurology and Therapeutics, *British Medical Journal*, April 22, 1893.

(1) In brain disease perverted function, whether mental or physical, must be attributed immediately to nerve-cell degeneration.

(2) Nerve cell degeneration may be primary or secondary.

(3) In general paralysis nerve-cell dissolution frequently occurs without the special morbid appearances usually considered as characteristic and in a degree causative.

(4) The balance of evidence is therefore in favour of a primary nerve-cell lesion being the essential morbid condition in this disease.

SYPHILIS OF THE SPINAL CORD.[1]

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THE *post hoc propter hoc* argument has been used so freely with regard to syphilitic diseases of the central nervous system that it seems eminently desirable to be able to make the diagnosis of cerebral or spinal syphilis without reference to the etiological factor. We should be able to recognise the symptoms of syphilitic disease whether or not the patient concedes, or we can prove previous syphilitic infection. My own experience during the past few years leads me to believe that this can be done with tolerable certainty in the case of the diseases under discussion.

Some time ago in a paper on "Multiple cerebro-spinal syphilis,"[2] I referred to the morbid lesions and to the important points of differential diagnosis for cases of syphilitic disease involving both the brain and the spinal cord. In this paper I propose to confine myself entirely to the diagnosis of spinal cord syphilis.

A fresh impetus has been given to the writings on this subject by Erb's short article[3] which appeared in the *Neurologisches Centralblatt* for March 15, 1892. With the same keen clinical insight which led to admirable results in his studies on muscular atrophies and of Thomsen's disease, Erb has sought to establish "a type" of spinal cord disease which he proposes to call "syphilitic spinal paralysis."

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[1] Read before the New York Neurological Society, April, 1893.

[2] *New York Medical Journal*, 1891.

[3] A detailed analysis of Erb's cases will be found in an interesting article by Dr. S. Kuh, of Chicago, published in the *Zeitschrift für Nervenheilkunde*, Bd. iii.

This special type is to be recognised by the following characteristics :

(1.) The usual symptoms of spastic paraplegia:
The peculiar gait, carriage and movements.

(2.) Marked exaggeration of the deep reflexes.

(3.) Muscular contractures slight as compared with the exaggeration of the reflexes.

(4.) Involvement of the bladder.

(5.) A slight, yet distinct disturbance of sensation.

(6.) Gradual onset of the disease.

(7.) A decided tendency to improvement.

We may concede at once that these points are well taken and that the symptoms stated above leave little doubt of the syphilitic nature of a given case. It is due to Rumpf to say that, on p. 351 of his well known monograph, he alludes to all these diagnostic symptoms in a case which was proved to be due to specific disease of the blood vessels in the spinal cord. He mentions spastic symptoms, paresis followed by paralysis of the sphincter of the bladder with incontinence of urine, exaggerated reflexes and contractures; later on in the course of the disease the diminution of sensation. The onset, too, was a gradual one. Rumpf, naturally enough, in speaking of his case which ended on the *post-mortem* table, does not refer to a tendency to improvement, and he does not insist, as Erb does, on the relatively slight muscular contractures as compared with the great exaggeration of the reflexes.

In developing his spinal type Erb recognises Rumpf's services in this field. The former may claim the special merit of attaching greater importance than Rumpf did to this difference between the intensity of development of muscular contractures and of increased reflexes.

The questions I have been trying to solve for some time past are—first, whether this Rumpf-Erb type represents the most frequent form of syphilitic spinal cord disease; and secondly, whether the cases that conform to this type represent *a* syphilitic spinal paralysis, or *the* syphilitic spinal paralysis, as Erb would have it. This is not a mere quibble of words. Much mischief was done, as every one will acknowledge, by Strümpell's procedure in describing the clinical features, of what he supposed to be polio-encephalitis, and in labelling these cases "*Die* cerebrale Kinderlaehmung," whereas it is now well established that if his cases exist at all they constitute a very small proportion of all the infantile cerebral palsies. I do not fear a similar mistake with regard to Erb's type of spinal cord disease, but there is a distinct tendency even now among more recent writers to imply that every case of syphilitic spinal cord disease should conform in most respects to the type described by Erb.[1]

This leads to the further question: Are the symptoms described by Erb so characteristic of spinal syphilis that we should trust to them entirely, or are there other symptoms which point with much greater force to the syphilitic nature of cord disease?

For the present the question is a purely clinical one, and one of great practical importance at that. The question in pathology can be brought up after the clinical question has been settled.

Let me record in condensed form a few cases, taken from private practice, of undoubted syphilitic disease of the spinal cord, from which we shall be able to draw the necessary inferences.

Case 1.—Mr. C., age 50, was referred to me by Dr. Gibney, September, 1891, and has been under my treatment ever since.

There was a distinct history of syphilis acquired two years previously, followed by constitutional symptoms.[2] He has been married 23 years; wife has had no children. He has been an active brain worker for many years, but moderate in his mode of life. In November, 1890, he had an attack of illness, during which his right eye-lid drooped; his left leg was paralyzed. In the course of a few weeks he recovered sufficiently to get about again. He also had "*eczema ani,*" and shortly after this was treated by various physicians for the tertiary symptoms of syphilis.

In the spring of 1891 he had a severe attack of influenza. A few months later in May, 1891, he developed a right hemiplegia, and a paresis of bladder and rectum. The left leg was still weak from the first attack. He recovered in a few weeks and was once more active in his business.

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[1] It is evident from Dr. Kuh's article (which has appeared since the above was written) that Professor Erb recognises mixed forms, &c., of syphilitic disease of the spinal cord.

[2] I am indebted to Dr. J. P. Tuttle for the early history of the case.

Two weeks before I first saw him he was compelled to go to bed again, his legs having grown weak and his bladder and bowels not acting voluntarily.

The condition I found him in was a rather pitiable one. In addition to complete spastic paralysis of both lower extremities, there was complete paralysis of both upper extremities ; there was no increase of the wrist and elbow reflexes, but a decided tendency to atrophy of the interossei particularly in the left hand. The knee jerks were enormously exaggerated; there were *extreme contractures* of the adductors of the thighs, and the very marked contractures of the posterior leg muscles inhibited the ankle clonus. The pupils were normal, speech normal; sensation in both lower and upper extremities was normal at the first examination. The electrical reaction of all nerves and muscles was entirely normal. There was retention of urine and inability to evacuate the bowels. This group of symptoms has been materially modified from time to time.

The involvement of upper and lower extremities with symptoms pointing to a very irregular invasion of the various systems of the spinal cord left no doubt in my mind as to the nature of the trouble, and in spite of very discouraging circumstances, I adhered to, and pushed, the mercurial and iodide treatment[1] to the extreme. During the first week the symptoms increased in severity. The paralysis became absolute and anæsthesiæ were soon associated with the paralysis. The anæsthesia was evident only in both feet and legs up to the middle of the thighs and over the sacrum and lower dorsal spine. There was no anæsthesia in the upper extremities, but very decided atrophy of the interossei. Within two weeks of the beginning of the treatment, while the conditions of the lower extremities remained unchanged, the upper extremities recovered completely and have been entirely normal ever since. Scarcely had these symptoms been recovered from before the intensity of the process in the lower cord seemed to increase. A bed-sore began to form which in spite of the most careful nursing increased in size until it was large enough to put one's fist in, and for fully eight months it was a threatening feature of the case, but finally healed completely under the surgical skill of Dr. Gerster. There has been a gradual improvement in the paralysis, so that after much patient treatment he is now able to walk about with the assistance of his crutch and an attendant, with every prospect of regaining the normal use of his legs unless a sudden relapse should set in. All anæsthesiæ have completely disappeared, the contractures are relaxed (the ankle clonus is now apparent), but spasmodic twitchings of the legs are still quite frequent. The patient has also recovered from several attacks of cystitis, but is still suffering from a specific ulcer of the rectum.

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[1] Inunctions of six grammes of mercurial ointment daily, and gradually increasing doses of the saturated solution of the Iodide of Sodium, from 10-150 drops, three times daily.

Recovery in this very severe form of spinal syphilis I consider fortunate enough, and doubt if it would have been possible if the symptoms had not been due to a specific process.

Please note that the contractures were extreme and that at one period very marked sensory disturbances existed.

Case 2 resembles the first in many particulars and still has run a very different course.

Mrs. X., a lady age 53, having five children, all healthy and married, was first seen in June, 1891. She was in good health until two years previously, and extremely active in spite of her weight of nearly 250 pounds. After the "blizzard," March, 1888, she had a troublesome cough and for a long time did not feel well. In spring of 1889 began to notice a heaviness in the left leg; this would remit at times and then would grow worse again, so that she was accused of having an hysterical affection. The trouble was restricted to the left leg for nearly two years, before the right leg became similarly affected. A few weeks before I saw her she was sent to Atlantic City by a physician who thought that the change of air would have a favourable influence upon her supposed functional paralysis.

The first examination revealed a very different state of things: (*a*) Extreme and absolute spastic paraplegia; not a muscle of a toe even could be moved. (*b*) Extreme contractures, so that the knees would be firmly clapsed together unless forcibly separated by pillows. (*c*) Violent spasmodic twitchings which gave rise to excessive pain. (*d*) There was complete anæsthesia of both legs as high up as a line passing two inches above the symphysis pubis. (*e*) Loss of faradic response without alteration of galvanic formula in all leg muscles and nerves. (*f*) No tendency to bedsores; and (*g*) absolutely no trouble with bladder or rectum.

The upper extremities were not affected. No history of traumatism or of unusual exposure, and, I beg you to remember, no history of an acute infectious disease. In the absence of these predisposing conditions—and more particularly on account of the extreme spastic symptoms associated with disturbances of sensation and with changes in electrical reactions (atrophy could not be made out on account of adispose tissue) without, however any interference with vesical and rectal reflexes, and no tendency to formation of bed-sores, I made the diagnosis of a specific myelitis. No other pathological process is as selective as this one. The symptoms pointed to involvement of the lateral columns, of the posterior roots or posterior gray matter, and also of the anterior horns or roots on both sides, and yet the column of Clark and the central grey matter were seemingly not affected.

I have persisted in specific treatment up to the point of tolerance with gratifying results, although the proof of any specific illness was a long time forthcoming. Some nine months after the beginning of treatment the lady's husband consulted me for very persistent headaches. Suspecting the nature of the trouble, I discovered not only that he had acquired specific disease many years since, but that he had been under a distinguished surgeon's care for bone syphilis only a year ago. The further progress of the wife's case has been marked by a gradual restoration of the electrical reactions, by total disappearance of the anæsthesiæ, by the cessation of all contractures and of the spasmodic twitchings, and by the slow and steady diminution of the spastic paralysis. She has regained moderate use of all the muscles of the lower extremities, but is still confined to her chair, as her legs will not yet bear her tremendous weight; but I expect her in a few months to learn to walk, as single efforts to stand have been successful, and she moves legs and feet very readily while sitting in a chair. I have not seen any but specific cases of spinal cord running such a chronic course and yet showing a tendency to improvement after the lapse of one or two years and even longer. I hold that in this case, next to the very remarkable improvement, the entire absence of bladder and trophic symptoms compared with the intensity in development of the other symptoms is the most characteristic diagnostic feature.

I could parallel this case by at least two others, but I do not wish to go into wearying details.

Case 3 is that of a colleague now practising in Hot Springs, Ark. He is 36 years of age; married, no children; formerly in good health; first symptoms of fatigue on slight exercise in 1880; began to look pale and run down. Was in Ottawa, Canada, at the time, and went to sea for a couple of years; did not improve, and he began to have rheumatic pains about joints, and suffered somewhat from dizziness. After his return he developed a very marked spastic gait which has slowly increased up to the present time; a slight irritability of the bladder, and loss of sexual power were symptoms from which he suffered at various times; but both these functions are now normal. He suffers most, however, from headaches. On further questioning I learned that one year ago after the onset of spastic symptoms he had some difficulty in speaking, associated with a numbness of the left side of the body; once had an attack of aphasia lasting 24 hours, and remembers a temporary weakness of left hand. As a result of my examination, I noted inequality of pupils, no other head or ocular symptoms; grasp excellent in both hands, but very exaggerated wrist and elbow reflexes; spastic gait with considerable rigidity, so that in walking the knees are pressed quite closely together; exaggerated knee jerks; no anæsthesia anywhere, no trophic symptoms, no tremors, no stammering speech, &c. I made the diagnosis of spinal syphilis, and then learned that fifteen years ago he had been inoculated with syphilis on the middle finger of left hand. I urged repeated mercurial and iodide treatment, but this has not relieved the spastic symptoms.

Please note again extreme spastic symptoms with contractures, slight involvement of the bladder, no sensory symptoms, and no atrophic symptoms, proving once more the selective character of the process. The case resembles in most respects those described by Erb as typical of syphilitic spinal paralysis.

Case 4.—This case, differing as it does from all other cases of spinal or cerebro-spinal syphilis I had seen, led me to seek for safe guides in differential diagnosis. I saw the young man, F. I., age 26, in consultation with Dr. Lustgarten, Oct. 14, 1892. He comes of a very markedly neurotic stock with the history of insanity in the family, and has one sister who is deaf and dumb.

Four years ago he had acquired undoubted syphilis. Since last spring has experienced difficulty in walking and in the use of his hands.

There was also the history and evidence of paralysis of the right superior oblique. At the first examination the young man was quite unable to lift himself up in bed; there was distinct atrophic paralysis of both trapezii, of infra and supra spinati, of deltoids and pectorals; a paresis of the entire arms and hands; grasp being almost nil.[1] All forms of sensation, particularly tactile and temperature sense, were normal. This was true of the upper extremities and of the lower extremities and on the trunk to the level of the umbilicus. Elbow reflexes could not be elicited. The lower extremities were much emaciated, more so the right than left; great weakness, scarcely able to lift legs an inch from the bed, and yet if put on his legs could stand a little; no ataxia, no absolute paralysis anywhere. Right knee jerk lost, left knee jerk very feeble; all superficial reflexes lost; no disturbances of vesical and rectal reflexes: no bed-sores, but a severe burn in the anæsthetic area on the right side just below the margin of the ribs.

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[1] At this first visit a careful electrical examination could not be made.

The diagnosis of wide-spread syphilis was clear to me, although the paralysis tended to the atrophic rather than to the spastic type.

The good results of treatment were very evident at my next examination about two months later, Dec. 6. Patient was able to sit up in bed and to walk about though feebly; the pupils now reacted to light, though sluggishly, and the muscles about the shoulder joint had recovered surprisingly from the atrophic condition. Normal sensation had returned, leaving a relatively small area of anæsthesia on the trunk. It is worth noting that at a still later examination the area of anæsthesia had again increased, while all other symptoms had receded.[1] Grasp of hands nearly normal, all muscles with the exception of the extensor groups in both forearms having been restored to their normal volume. Faradic responses diminished but present in all muscles even in the extensors, with the exception of the anterior tibial group of the right side. Both knee jerks can now be elicited by reinforcement, and his mental condition has much improved.

Four months after first examination patient was able to call upon me at my office, improved in every respect, walking without assistance and simply using a walking-stick to get about with. He was still affected with double vision, in consequence of paralysis of right superior oblique.

If we except the slight ocular trouble, this was a spinal case clearly enough. The paralysis was of the atrophic and not of the spastic order. The involvement of both the upper and the lower extremities; the unequal development of the symptoms (the paralysis and anæsthesia being very pronounced, while the bladder was not involved, and the electrical conditions and the reflexes were less distinctly altered, and trophic disturbances were wanting entirely), this peculiar and unequal development of symptoms, as well as the rapid recovery, proved the case to be one of spinal syphilis with slight cerebral involvement.

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[1] Drawings illustrating this variation in the areas of anæsthesia were exhibited at the meeting.

In the cases just recorded the diagnosis of spinal cord syphilis can hardly be questioned. I share the fate of other writers, in not being able to offer *post-mortem* evidence of these diagnoses. Recent syphilis of the central nervous system if properly recognised should not lead to *post-mortem* examinations, excepting in rare instances; and it is true, moreover, of all syphilitic diseases that the clinical manifestations are in many respects far more reliable than are the results of histological examinations of the diseased tissues, for, curiously enough, the pathologist in making his diagnosis has to rely very largely upon points obtained by clinical examination.

Now what were the features which led to the diagnosis of syphilis in the cases I have detailed? In one there was wide-spread atrophic paralysis, in three of them there was spastic paraplegia of the most pronounced type. In these the reflexes were excessively exaggerated. In two of them the muscular contractures were slight; in one of them they were extreme. In one case there was permanent involvement of the bladder. In the others no involvement. In all but one of them sensation was disturbed. In two the onset was gradual. In the others it was quite sudden. All of them have shown a decided tendency to improvement.

I have mentioned first the features in which they conformed to Erb's type, but in other and equally important, if not more important, points they differ from that type. In one instance there was a distinct atrophic paralysis, with all the symptoms that pointed to a wide-spread affection of the gray matter of the cord, that would have reminded one of a poliomyelitis, with the exception that, as predicted on the strength of the syphilitic character of the disease, the patient made a very rapid recovery.

In Case 1, in which the diagnosis of syphilitic disease is to my mind beyond dispute, the contractures were extreme: the bladder has remained involved up to the present time, and bed-sores formed as in the most typical case of acute transverse myelitis.

These points of difference did not conceal the true nature of these cases, and I beg of you now to consider a few facts which have impressed themselves upon my mind as more characteristic of spinal cord syphilis than any other I have been able to fasten upon.

(1) The most striking of all is the unusual distribution of the disease over the greater portion of the cord, involving as it did in several cases the cervical, dorsal and the lumbar enlargements.

(2) The relatively slight *intensity* of the morbid process as compared with the *extensive area* involved, as evidenced by the preservation of some of the functions of the cord with complete loss of others.

(3) A rapid dwindling of some of the symptoms, and the very chronic persistence of others. In several of the cases, for instance, the anæsthesia lasted but a very short time, while the paralysis was recovered from with extreme slowness.

(4) The very frequent history of other symptoms pointing to specific disease in the same or other parts of the central nervous system.

I shall be inclined to suspect specific disease of the cord if the patient presents symptoms of paralysis, whether they be of the spastic or flaccid character, and whether the contractures be slight or not, provided he furnish evidence of a morbid process affecting a very large part of the cord, and yet showing a relatively slight intensity at any given level of the cord; if, for instance, he exhibit the symptoms of extreme paralysis, spastic or atrophic, with partial or slight anæsthesia of the parts paralyzed, with little or no involvement of the bladder, and particularly if we can find traces of specific disease in other parts of the central nervous system.

These cases remind one more or less of those which were formerly diagnosticated as irregular cases of subacute or chronic myelitis; and if we refer for a moment to the nature of the morbid process we can readily understand why this irregularity in the development of the symptoms should be the most characteristic of all of the diagnostic features of these cases. There is not in them as in the cases of acute myelitis a morbid process which is rapidly destructive and which rapidly advances across the entire cross section of the cord, involving all the symptoms due to loss of function of the various spinal systems. If the syphilitic disease be the result of a specific endarteritis of the vessels of the cords, we know that some and by no means all of these vessels are affected, and that the disease advances slowly from one group of vessels to another. If there be diffuse specific infiltration it also invades but very slowly one part after the other. It has a remarkable tendency, too, to increase for a time and then to recede, whether as a result of treatment or not, and then possibly to increase with renewed force. If the infiltration start from the meninges it invades most frequently the lateral columns first, often at symmetrical points, and advances very slowly from white to gray matter. The intensity of the process is spent often upon the lateral columns, hence the frequency of the spastic symptoms. It may invade the gray matter, giving rise to sensory symptoms, sometimes to atrophic symptoms. It is very apt to recede from the gray matter, restoring sensation, the conditions of nutrition and the normal condition of the bladder; but it seems to halt at the lateral columns, leaving the spastic symptoms intact for a very long time.[1]

If we try to harmonize the clinical symptoms with the pathological conditions it would seem much more natural to lay greater stress upon those symptoms which are due to the peculiar retrograde features of the morbid process, than upon those symptoms which are dependent upon the area attacked by the disease.

For all these reasons it seems better to fasten upon *the relatively slight intensity of the disease,* and consequently of all symptoms, *as compared with the extensive area involved,* and upon the *tendency to remissions and relapses* than upon any mere association of symptoms due to involvement of the different parts of the cord at any one given area. I do not mean to dispute the existence and the propriety of establishing Erb's type of spinal disease, but I do believe that if we do not regard other and broader points of diagnosis we shall often fail to recognise spinal cord syphilis.

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[1] In a paper entitled "Tabes and Syphilis" (not yet published), read before the American Neurological Association, July, 1893, I have laid particular stress upon the anatomical features of this process.

ON DIABETIC NEURITIS, WITH A CLINICAL AND PATHOLOGICAL DESCRIPTION OF THREE CASES OF DIABETIC PSEUDO-TABES.

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SINCE Marinian and Bouchard (1) first pointed out, in the year 1884, the fact that the knee jerks were frequently absent in cases of diabetes mellitus, the attention of medical observers has been especially drawn to the various nerve troubles and complications which not uncommonly arise during the course of this malady.

Buzzard in 1886 attributed the loss of the knee jerks in some cases to peripheral neuritis. Negative pathological evidence in support of this view was also obtained through the researches of Rosentein (2), Niviere and others. In the cases reported by these observers, the spinal cord when microscopically examined was found to be healthy.

Clinically Leyden (3) has divided the neuritis of diabetics into three groups, *i.e.* :—

(1) The hyperæsthetic or neuralgic form, in which there is more or less severe pain. V. Ziemssen first ascribed the various neuralgias which not uncommonly occur in diabetes to be the result of neuritis. These neuralgias are not infrequently symmetrical in their grouping.

(2) The motor or paralytic form. In this variety there is paralysis of varying degree of the muscles of the legs, or of single muscles, or groups of muscles.

(3) The ataxic form—pseudo-tabes. Here there is ataxia, and various sensory symptoms, such as numbness, formication of the feet, &c. The knee jerks are lost.

Whilst agreeing with Leyden that cases of diabetic neuritis may to a large extent be divided into these three different groups, I believe the clinical picture would be truer and more complete if we restricted them to two fairly distinct varieties, *i.e.*—

(*a*) The motor or paralytic form.

(*b*) The sensory or ataxic form.

In this latter variety I would include Leyden's hyperæsthetic or neuralgic group, believing that the symptoms characteristic of this class do in many instances develop into more serious nerve complications. In this group the sensory, vaso-motor and trophic nerve fibres are chiefly affected, and the clinical symptoms presented bear a very close resemblance to those of locomotor ataxy.

In the *British Medical Journal* of March, 1887, I drew attention to those cases of diabetes in which there was not only loss of the knee jerks, but also a certain degree of ataxia, and ascribed the symptoms to a somewhat widespread peripheral neuritis. To this type of case the term diabetic ataxia, or diabetic pseudo-tabes, may I think be very suitably applied. In the same year Dr. Pavy (4) in his address on diabetes at the Washington International Medical Congress described a similar class of case.

"He stated that this form of ataxy occurs in persons past the middle period of life, that they complain of pains of varying character in the limbs, and that the ataxy is not exactly the pure form met with in tabes dorsalis. For the most part these persons can stand and maintain their balance fairly well with the eyes shut, but they walk with difficulty. They cannot properly maintain their balance. Associated with these symptoms there is some anæsthesia and hyperæsthesia, and various forms of paræsthesia. They often feel as if they were walking on pebbles. The disease is most marked in the lower extremities; sometimes, however, the upper extremities are also affected. The patient complains of an aching of the bones at night. The condition appears to be due to peripheral neuritis."

To this vivid description of the symptoms presented by cases of diabetic ataxia I would only add that the knee jerks are diminished or frequently absent, and that these patients are occasionally liable to vaso-motor and trophic troubles, as evidenced by œdema, erythema, and perforating ulcers of the feet.

Death may also result from gangrene of the extremities. There are no doubt cases of diabetic neuritis which present motor and sensory symptoms; but in the main I think their division into motor and sensory varieties is a correct one.

Some twenty cases of diabetic neuritis have been recorded during the last few years, notably those by Leyden, Charcot (5), Buzzard (6), Althaus (7), Auché (8), and Eichhorst (9). They have varied much in their symptoms; in some the motor and in others the sensory nerves have been chiefly affected. As our knowledge of the disease increases I opine that it will be found that the sensory nerve fibres are especially liable to be affected in diabetes.

Although it will be seen from what has already been stated, that clinically diabetic neuritis has been abundantly recognised, very little pathological proof of this lesion has been forthcoming. Indeed, with the exception of a case published by myself (10) in the *Lancet* of July, 1887 (an abstract of which follows), and of the observations of Auché in 1890, and Eichhorst in 1892, I have been quite unable to find distinct pathological evidence of this form of neuritis.

I trust that the cases appended will add still further to our pathological and clinical knowledge of this disease. They belong to the sensory or ataxic group, and may very properly be termed cases of diabetic pseudo-tabes.

Case 1.—A. D., aged 56. Diabetes. Ataxic symptoms, perforating ulcers of both feet; coma; death.

Frame work knitter and hawker; was first seen on May 27th, 1886. For eighteen months past he had passed large quantities of urine, and had suffered from great thirst. He was never of temperate habits, and was addicted to "drinking bouts."

No history of gonorrhœa or syphilis could be elicited, but strong presumptive evidence of venereal taint was present in the form of two scars, one in each groin, and various suspicious-looking marks on both legs. Eight months previous to admission he complained of pains in the legs and stated that his feet felt dead.

At Christmas, 1885, he caught cold and suffered from an aggravation of the foot symptoms, together with abdominal pain and vomiting. He was described at this time as walking on his heels. In February, 1886—three months and a half before his death—the ulcers of the feet first appeared.

When first seen by me the patient was suffering from bronchitis and symptoms of commencing diabetic coma. When supported he could, however, manage to walk across the room. His gait was distinctly ataxic. The arteries of the upper limbs were evidently diseased. The pupils reacted feebly to light. There was diminished cutaneous sensibility of both feet, and the lower thirds of both legs. The knee-jerks were totally abolished. The skin over the legs and feet was of a livid hue, and below the normal in temperature. A fœtid odour emanated from both feet. The ulcers commenced as corns. The largest ulcer, of the size of a two shilling piece, was situated over the plantar aspect of the meta-tarso-phalangeal joint of the right great toe. An ulcer of the same character but of smaller size was situated over the plantar aspect of the head of the fifth metatarsal bone of the opposite foot. Carious bone could be felt in each case. The patient's urine was acid; sp. gr. 1030, contained sugar and a trace of albumen.

He died of diabetic coma on the fourth day after coming under my observation. There was no history of gout, rheumatism or injury in this case.

The spinal cord, and the nerves of the right lower limb were examined. The naked eye changes which were detected were (1) considerable enlargement of the lower third of the posterior tibial nerve. The nerve was surrounded by inflammatory material, in which was embedded the atheromatous posterior tibial artery; (2) the plantar terminations of the nerve were discoloured and diminished in size.

The cord and peripheral nerves were examined by Mr. Bowlby of St. Bartholomew's Hospital, who demonstrated the following changes. In the lumbar region of the cord there was marked atrophy of the cells of the grey matter, which were granular and had lost their processes. In the dorsal region the changes were even more marked. There was no cell exudation in either the white or the grey matter. In the peripheral nerves the changes were most evident in the posterior tibial trunk, where the white substance of Schwann was replaced by granular material which did not stain black with osmic acid. Some of the axis cylinders were normal, but many were atrophied and the subject of fatty degeneration. The anterior tibial nerve showed a similar degeneration, but in a lesser degree, and in the sciatic the bulk of the fibres were normal. There was also a slight increase of the connective tissue basis.

Dr. Byrom Bramwell of Edinburgh also described similar changes in the peripheral nerves of this case, together with well marked vascular disease involving not only the larger vessels, but spreading also to their minute subdivisions. There was no ataxic lesion in the spinal cord.

Case 2.—Charles B., aged 72. Diabetes. Ataxic symptoms; gangrene; death.

This case came under treatment four years before death took place. For ten years previous to this date he had suffered from double sciatica, and for some years pain of a lightning and burning character had been especially marked in both lower extremities.

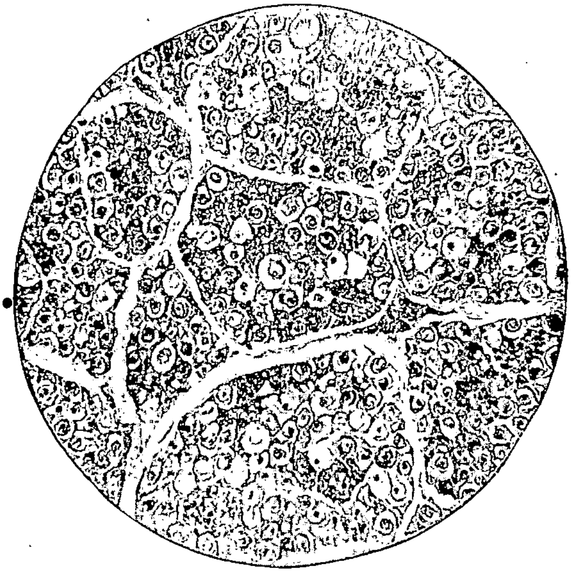
When first seen he complained greatly of the above-mentioned pain, its seat of greatest intensity being in the left foot and leg. Both knee-jerks were diminished, and could only be obtained on reinforcement. There was marked muscular inco-ordination in the dark or with closed eyes, but no evidence of paralysis.

Anæsthesia was present to a varying extent in the toes of both feet. To a lesser degree this anæsthetic condition was also present in the feet and lower thirds of both legs. He frequently complained of burning and pricking sensations; most of the teeth had fallen out in a fairly sound condition. The pupils reacted to light and accommodation.

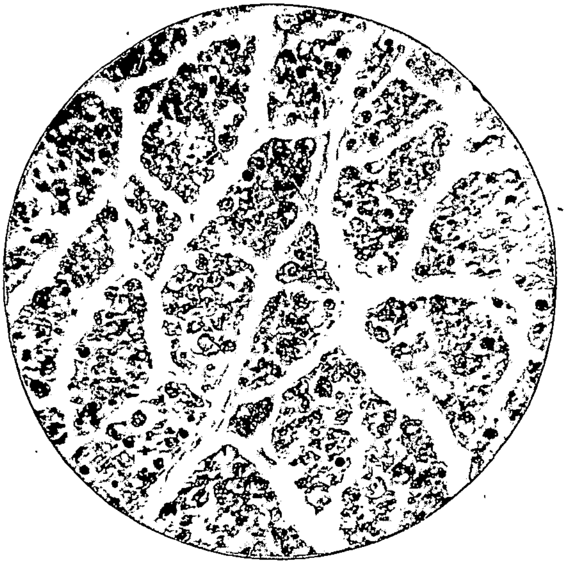
He suffered from great thirst and passed large quantities of urine, which was acid, sp. gr. 1025, and contained sugar. There was no history of alcoholism, syphilis or rheumatism.

This patient occasionally suffered from a peculiar erythematous and œdematous condition of the feet, which appeared to be distinctly co-incident with an increase in the amount of sugar passed in the urine. During one of these attacks of "erythematous œdema" a small ulcer formed on the plantar aspect of the left great toe. This ulcer was surrounded by a very marked anæsthetic zone. The urine during this attack increased in quantity, sp. gr. 1030, and contained much sugar.

The course of the malady in this case was varied, but pain was always a constant and distressing symptom. For some time before death the loss of the knee-jerks was complete. He died of gangrene of the left foot and lower third of the left leg in July, 1891.



Healthy Nerve. Weigert stain.



C. B. 72. Sciatic, showing increase of connective tissue basis. Weigert stain.

The gangrene commenced in the ball of the left great toe (the site of the previous ulcer), and gradually extended upwards. It was of two months' duration. The anterior tibial, posterior tibial and sciatic nerves all showed a degeneration very similar to that described in the first case. These changes were most marked in the posterior tibial trunk. There were most distinct arterial changes, consisting of atheroma of the large artery, and thickening of the coats, and in some instances of blocking up of the smaller microscopic blood vessels.

I am much indebted to Dr. W. B. Ransom for the microscopic examination of the nerves in this and the following case.

Case 3.—William A., aged 62. Diabetes, ataxic symptoms, gangrene, death.

This case was under observation for a period of nearly four years, October, 1887, to May, 1891. When first seen he gave a history of having suffered from great thirst for more than two years. During this time he had also passed an excessive amount of urine. He complained of shooting and burning pains in both lower limbs and feet, but more especially in the right foot. Cutaneous sensibility was diminished in both feet.[1] Both feet were extremely painful, and presented the condition of "erythematous œdema" already described in the foregoing case.

There was slight muscular inco-ordination; no paralysis. The knee-jerks were diminished but not totally abolished; the pupils reacted to light and accommodation; urine, sp. gr. 1036 and contained sugar but no albumen or peptone.

The symptoms in this case (although varying from time to time) gradually became aggravated until about four weeks prior to his death gangrene commenced in the right great toe and extended up the foot to the junction of the lower with the middle thirds of the right leg. Mortification also set in, in the left toe and foot, but did not extend to the leg. Death was due to exhaustion. For some months before death the knee-jerks could not be obtained.

The sciatic, anterior and posterior tibial nerves were examined and showed marked parenchymatous degeneration, the changes being very similar to those found in the nerves of the foregoing cases. Vascular disease was also present in the form of atheroma of the larger arteries, and of thickening of the walls and blocking up of the lumen of the smaller blood-vessels. There was no history of alcohol, syphilis or rheumatism in this case.

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[1] This refers to the condition of the feet generally, but not during an attack of erythematous œdema.

insufficient to explain all the clinical and pathological facts of cases such as the above.

Here we have other factors at work. In the first place there has been long continued malnutrition, and in the second there is found extensive vascular disease.

Whilst I do not deny that a specific toxic poison may not have had a share in the causation of the neuritis in these cases, I would at the same time ascribe the disease mainly to vascular disease and malnutrition. Vascular changes were apparently as much a part of the morbid process as peripheral nerve degeneration.

The posterior tibial artery is perhaps the one most liable of all the arteries of the body to atheromatous degeneration, and it is in the posterior tibial nerve trunk supplied by branches of this vessel that the nerve degeneration was especially marked.

The condition of erythematous œdema is apparently very closely allied to the vaso-motor neurosis described by Weir Mitchell under the name of erythromelalgia.

The treatment of these cases resolved itself into the treatment of diabetes and the maintenance of the general health.

Morphia and antipyrine were of great service in allaying the constant pain. Operative interference would, I believe, only have acclerated death.

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Note on cases in which the nerves have been examined microscopically:—

(1) EICHHORST. (*a*) Woman, 45. Death due to coma. No paralysis, knee-jerks abolished, no loss of sensation, no sign of ataxy; duration two years. (*b*) Woman, 23. Death due to coma. No history; knee-jerks absent; muscular weakness of the limbs.

(2) AUCHÉ. (*a*) Paresis of legs, muscular cramps, gangrene of right foot. (*b*) Itching, tingling of feet, legs, and hands; knee-jerks absent. (*c*) Cramps of calves, diminished sensation on dorsal surface of forearms, subunguinal hæmorrhage and shedding of the nails.

Since receiving the proof of this paper I note that Leyden *(Deut. Medizin. Zeitung*, June 5th, 1893) refers to a case of diabetes with renal disease, in which not only lesions in the peripheral nerves but also in the spinal cord were present. The disease in the cord was not limited to any system. *British Medical Journal*, June 24th, 1893. Epitome, Current Literature.

Clinical Cases.

A CASE OF INTRA-CRANIAL HYDATID TUMOURS WITH HEMIPLEGIA, AND WASTING OF THE PARALYSED LIMBS.

BY J. MICHELL CLARKE, M.A., M.D., M.R.C.P.
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The patient was a man aged 29, who complained of intense pains in the left temple, of failure of sight in the left eye, and of loss of power in the left arm and leg. He had formerly been of intemperate habits, but during the last three years had been very abstemious. With the exception of syphilis six years previously, he had had no previous illness.

For 18 months he had suffered from occasional epileptiform fits, in which consciousness was lost and the tongue bitten. The weakness of the left side had been coming on for 12 months. Twice during the past six months he had had attacks of pain in the left eye with double vision.

Three weeks before admission he was seized with violent pains in the left temple, radiating over the left side of the head to occipital region; these pains have never left him since then.

He was admitted on January 21, 1893. He was very restless, groaning continually on account of the great pain in his head. He seemed dull and drowsy, but could not sleep, and took no notice of what was going on around him; it was difficult to get a connected account of his illness from him, because of the mental hebetude. He could walk, but only feebly, and dragged the left leg.

On examination the wrinkles of the left side of the forehead were somewhat smoothed out, he could screw up the right eye more tightly than the left, and on smiling the mouth was drawn to the right. Acting separately both angles of the mouth could be well moved. Localisation of sensations, tactile and pain sensations, were impaired over the left side of the face; thermal sense normal. He protruded his tongue straight. There was incomplete left ptosis; the left pupil was larger than the right, and did not re-act to light. There was optic neuritis of moderate intensity in each eye. There was decided loss of power in the left arm and leg; with the dynamometer the right grasp being 120, the left nil. The power of localising sensations was distinctly impaired over the left side, otherwise sensation over the trunk and limbs was normal. The thoracic and abdominal organs were healthy. There was incontinence of urine, which contained no albumen. The left knee jerk was exaggerated, but there was no ankle-clonus.

On *Jan.* 28, left ptosis more marked, the left external rectus was now paralysed, the optic neuritis had increased, but he had regained some power in the left arm and leg, the grasp on this side being 29.

On *Feb.* 14 the left eye was completely paralysed and immovable, there was cycloplegia and nearly complete loss of vision. Right eye: movement good, pupils acted well; V.=J. 10. He had some convulsive twitchings of the left hand. The intense pain continued and prevented sleep.

On *Feb.* 28, paralysis of the *right* internal rectus and loss of vision over the nasal half of the *right* visual field was noticed. With the left eye he could not detect a ray of light thrown into the eye from the ophthalmoscopic mirror. Muscular wasting in the left arm and leg had been noticeable for some time. Measurements gave R. thigh 17 inches in circumference, L. 16 inches; R. calf 11½ inches, L. calf 11 inches; R. forearm 9½, L. forearm 7½ inches. The electrical reactions, taken on two occasions at a week's interval, showed marked reduction of faradic irritability of muscles of left arm and leg. With the constant current there was increase of irritability. Over the peroneal nerves a contraction was obtained with a current of 8½ milliampéres on the right, and 7 milliampères on the left side. There was a similar difference between the two sides over the ulnar nerves. The muscles of the left forearm and leg reacted more readily than those on the right side, and further in the former A C C > K C C, K C C = A O C. There was thus qualitative reaction of degeneration.

From *March* 4 to 28 a remarkable degree of improvement in the paralytic symptoms occurred. The patient could open the left eye, move the left arm freely, and even feed himself. The mental condition, however, grew steadily worse. He had a convulsion about this time in which the muscular twitchings began in the left hand, spread next to the left side of the face, and then became general.

On *April* 1 he became very much worse, lay in a stuporous condition, and could not masticate his food. There was now rigid spasm of the left arm and leg. On the 3rd loss of sensation over the left eyeball was noticed, and there was very marked unilateral sweating on the right side of the face, head, and trunk. The head and eyes were turned to the right, and the right pulse was distinctly larger than the left. He died in coma on the 5th; the temperature, which had never exceeded 99° during the progress of the case, reaching 105° before death. The treatment consisted of the administration of iodide of potash in large doses, with mercurial inunctions, and hypodermic injections of morphia for the relief of pain.

At the autopsy a large hydatid cyst, containing several daughter cysts, was found in the posterior part of the left cerebral hemisphere. The cavity measured three inches in the antero-posterior, and two inches in the vertical and transverse diameters respectively. Its walls were smooth and rounded, and showed no signs of inflammatory changes. It thus occupied and replaced the greater part of the white matter of the parietal and occipital lobes. It came nearest to the surface above, under the anterior part of the superior parietal, upper and upper-mesial portions of the ascending parietal convolutions. In this situation the pia mater was firmly adherent to the cortex, tearing it on removal; the grey matter of the cortex was reduced to a layer about $\frac{1}{12}$ inch in thickness. The cortex had undergone most extensive atrophy under that part of the ascending parietal convolution which is concerned in the movements of the arm, and here the ascending frontal convolution was also encroached upon for a short distance. The boundaries of the cyst were sharply defined and surrounded, in all other parts than those first mentioned, by healthy white matter; a broad layer of healthy white substance intervening between it and the cortex.

Another cyst, the size of a large hazel nut, was found at the back of the left orbit, lying between the anterior clinoid process and the sella turcica, just posterior to the sphenoidal fissure and optic foramen, and had compressed and destroyed the left optic nerve and the motor nerves to the left eyeball. All other parts of the brain were healthy. Other cysts were present in the spleen and kidneys.

Unfortunately the extreme upper part of the cervical region of the spinal cord was not reserved for examination, but a large number of sections of other regions was made, stained in carmine, aniline blue black, and by the Weigert and Pal-Weigert methods, and carefully examined microscopically.

A well-marked degeneration of the left crossed pyramidal tract extended throughout the whole length of the cord. The nerve fibres had largely been destroyed, the sclerosis was of moderate intensity. Sections stained in carmine showed numerous spider cells.

On carefully comparing the two sides in a number of sections the motor cells of the anterior cornu were less in number on the left than on the right side, some had undergone atrophy, others appeared granular and swollen, with loss of their processes. On the left side also more cells were pigmented, and there was a larger amount of pigment in cells thus affected, the nucleus being often obscured. The vessels in the left cornu were more injected, and the peri-vascular sheaths were distended. In some sections there appeared to be a comparative diminution in the fine myelinated fibrils of the grey matter in the left anterior cornu. These changes were more evident in the lower cervical and upper dorsal regions than in the lumbar and sacral parts of the cord. In some sections these alterations in the left cornu were slight, and in most individual sections could be easily overlooked, it was only by comparing the two cornua in a very large number of sections taken from different levels that it became apparent that there was an evident though partial change in the motor cells of the left cornu throughout the cord. When compared with the right side sections of some of the left anterior roots showed disappearance of some nerve fibres. I regret that the examination was not rendered complete by the preservation of some of the smaller peripheral nerves.

Remarks.—Hydated tumours of the brain are rare in this country. Dr. Thomas, of Adelaide, states (*Hydatid Disease*, Adelaide, 1884, p. 124) that 5 per cent. out of a total of 1,987 cases of hydatid cysts were connected with the central nervous system. They give rise to no characteristic symptoms.

In the present case the diagnosis was clear from the localizing signs, first, of a tumour at the back of the left orbit, secondly, of another, and almost certainly larger growth pressing on the motor centres, chiefly on the arm and hand centres, of the right cerebral hemisphere. The growths were supposed to be syphilitic in nature, and from the usual tendency of syphilitic growths to invade the cortex from without (dura mater), it was thought probable that the tumour arose in this situation. The syphilitic nature of the growths was rendered probable from the fact of the history of previous syphilitic infection, and from the rapid improvement which at first took place under treatment. Further, as multiple intra-cranial growths are practically met with most often as a result of syphilis or tubercle, the absence of any family history of tuberculosis was taken into consideration. An operation was not advisable in the presence of more than one tumour.

A point of interest is the occurrence of wasting and the qualitative reaction of degeneration in the muscles of the hemiplegic side. As is well known in the immense majority of cases of hemiplegia, no such changes occur. When they do occur they are generally considered to be due to some specially irritative influence propagating itself along the descending pyramidal tracts to the motor cells of the anterior cornu. This irritative influence is exemplified in this case by the destructive changes which the cyst had excited in the motor cortex, especially in the part related to the arm and leg centres. The appearances described above show that the muscular changes were due to alterations in the motor cells. The morbid alterations, though slight, are in the whole series of sections quite unmistakeable. Changes in respect of pigmentation have been mentioned above in addition to the more striking signs of atrophy of cells. It is often and justly insisted upon that pigmentation of nerve cells is normal, varying greatly in degree in cells of different parts of the central nervous system. But when the cells of one side of the cord in large numbers contain much more pigment, often to the extent of almost occupying the entire protoplasm of the cell, than those of the opposite side, I think we are justified in regarding it as an abnormal change. As to what part vaso-motor changes may have played in the condition of the cells observed, it is not possible to say. Another point of interest is the total absence of rigidity in the affected limbs until three days before death; one month before death the knee-jerks could not be obtained; they were only, however, tested in the lying posture. Thus, in the muscular wasting, in the altered electrical reactions, and in the flaccidity of the limbs the hemiplegia in this case differed from that of the ordinary type. These differences are accounted for by the changes in the motor cells of the anterior cornua. Whether we may attribute the morbid condition of these motor cells to the destructive changes taking place in the motor convolutions is more doubtful, but it is interesting to note as pointing to a connection between the two conditions, that the motor convolutions were most affected over the arm and trunk centres and the left anterior cornu in the lower cervical and upper dorsal regions. The connection if a causal one cannot be direct, but is probably an interference of a reflex nature with nutritive processes in the spinal cells.

The distinct loss of the power of localization of sensory impressions in the left arm and leg apart from any other affection of sensation is in accordance with recent experiments on the cortex cerebri.

DIPHTHERIAL PARALYSIS—ALLOCHEIRIA.

BY WILLIAM GAY, M.D., M.R.C.P.

THE following case is chiefly of interest on account of the occurrence of a rare nervous symptom, which, as far as I am aware, has not hitherto been observed in the course of diphtherial paralysis. It consists of a perversion of common sensory impressions to such an extent, that they are localised to an exactly corresponding area on the opposite side of the body. This symptom was first prominently noticed by Obersteiner, who suggested that the condition should be called sensory allocheiria or shortly allocheiria (ἄλλος χέιρ). Such a term is rather misleading, as it might at first sight be imagined to refer to the hands alone, whereas the symptom is most often discovered in the legs.

I have to thank Dr. Christie Wilson, of Doncaster, for handing the case over to me, and allowing me to publish it. M. A., aged 13 years. Father and mother alive, and always in good health. One brother and one sister living and in sound health. No hereditary neurosis. No personal neurotic history at all. The child had just left school to work in a mill, and had always been very strong and healthy. Eleven weeks before I saw her, Dr. Wilson had attended her for a very severe attack of pharyngeal diphtheria, and her condition was regarded as hopeless. She, however, gradually recovered from her original illness, but it was noticed four or five weeks from the commencement that fluids regurgitated through her nose, and that her once shrill voice was reduced to a sort of falsetto whisper. Soon her near vision became deranged and diplopia supervened. The pupils were noticed by Dr. Wilson, widely dilated. There was at this time no actual paralysis, but a generally distributed weakness, so that she was unequal to any exertion and stumbled about in walking. Present condition. Patient is a well developed and healthy looking girl, and makes no complaint except of lassitude. On walking towards me, however, she had to rest her hands on various objects to support herself. Romberg's symptom extremely well marked. She would actually have fallen, standing with her feet together and eyes shut, if I had not caught her. There was considerable ataxia of all four extremities. No knee, wrist, triceps or jaw jerk. No superficial reflexes. All muscular efforts weak and ill-sustained. Special senses: Eyes—accommodation still defective; pupils dilated, but respond to light and accommodation; weakness of internal recti; otherwise normal. Hearing normal. Smell gone, this was noticed soon after the defect in voice. Taste not tested; but the fact that she could distinguish between some kinds of food, shows that it was probably normal. Common sensation—slight touches not felt at all, but firmer ones referred to exactly corresponding points on the opposite side of the body. This was tested again and again, and created such astonishment in the patient that she would not credit my observation, until corroborated by her parents. She was then astounded to find, that upon taking my hand from her eyes, she had localised a touch upon her ear, face, tongue, arms, and toes, to the opposite side of the body. On only one or two occasions did she hesitate for a moment in so localising her impressions. There was no delay in the transmission of sensory impulses, nor any polyæsthesia. No paræsthesia. Discrimination of heat from cold correct, although there was some slight hesitation in detecting finer shades of difference. Painful sensation not tested. The muscular sense of all four extremities entirely gone. Her limbs were put into all sorts of grotesque attitudes, but the only thing recognised was the warmth of my hand. Movements of head and trunk correctly localised. Voice at present would be best described by the term "cracked," and had by no means regained its old power. Unfortunately, she could not be persuaded to allow a laryngoscopic examination. The soft palate was only slightly paretic, for the b of rub, rub, rub was pronounced like rub, rub, rubm. The condition of affairs was evidently rapidly improving, and in about three weeks from the time I first saw her, she had, but for a feeling of weakness and the loss of knee-jerks, lost all her symptoms.

I was extremely unfortunate in not seeing the case at an earlier period, when all the symptoms were more pronounced. That the essential nature of the affection was diphtherial paralysis can hardly be doubted. The only question that arose was, whether hysteria, which sometimes supervenes, or gets tacked on the symptoms of diphtherial paralysis, was in any way responsible for certain phases of the case, *e.g.*, the aphonia, anosmia and disorders of common sensation. I think not. There was nothing in the hereditary or personal history of the child to favour such a suggestion, and although some of the symptoms are distinctly unusual in the course of diphtherial palsy, they are with the exception of the transferred sensation, not unknown. I regard the case, therefore, as an instance of diphtherial paralysis, pure and simple.

Laryngeal paralyses are uncommon, but by no means unheard of in diphtherial paralysis. What, however, makes me regret the absence of a laryngoscopic examination the more is that the patient's voice was identically similar to that of a man, a tabetic, who was the subject of ataxia laryngis, and who supplied me with the text for a paper[1] upon that affection, read before the Neurological Society. It will be noticed that there was ataxia of all the extremities, and a certain parallelism between diphtherial paralysis and tabes dorsalis will presently be pointed out. Anosmia is another of the infrequent symptoms met with in this case. It might presumably have been the result of nasal diphtheria, of which, however, there was no evidence. It must, therefore, have been a purely sensory symptom, and equally uncommon in both of the above-mentioned affections.

The most interesting symptom present was, of course, the allocheiria, of which I have been unable to find any example in English literature, although, doubtless, some exist. Obersteiner relates four cases of his own, and five from other German sources, but in no instance was the symptom so widespread or definite as the above. All of them but one, which was hysterical, occurred in the course of tabes dorsalis, which has a close relationship with diphtherial paralysis. Notice, *e.g.*, the loss of knee-jerk at one end of the nervous system, co-existing with ocular symptoms at the other end, the ataxia, loss of muscular sense, &c. The occurrence of allocheiria in the course of diphtherial palsy need therefore excite no surprise. The condition is generally associated with considerable sensory disturbances and loss of muscular sense. The coincidence of these symptoms is best explained by the view entertained by Gowers,[2] that the fibres conveying muscular sensibility correspond with the non-decussating fibres of the postero-median column, with which are mixed decussating fibres from the posterior commissure and neck of the posterior cornu, conveying tactile sensibility. The correct appreciation of temperature is probably explained by the theory that the nerve fibres conveying these impressions are situated in the lateral columns near the antero-lateral ascending tract, which probably conveys painful sensations. According to this view, the discrimination of heat from cold and the appreciation of painful impressions are conducted by non-decussating fibres.

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[1] BRAIN, vol. x., p. 494.

[2] "Diseases of the Nervous System," vol. i., p. 133.

It is extremely difficult to offer any suggestion concerning the pathological state, which gives rise to the condition, allocheiria. Several recent observations go to show that a very intimate connection exists between corresponding sensory areas of the two sides of the body, both physiologically and pathologically. Dumontpellier, for example, by ætherising one arm in a particular position, was able to open painlessly an abscess situated in a corresponding area of the opposite side. Recognising this close sensory relationship between the two sides, may one venture to throw out as a possible hypothesis that, under certain circumstances, when the decussating fibres, together with the non-decussating fibres conveying muscular sense impressions, are, for some reason, put out of court, the remainder of the non-decussating fibres take on the additional duty of transferring common sensory impressions, with which, under normal circumstances, they have nothing to do. If this were so, such impressions would reach the brain on the same side, and would therefore be referred subjectively to the opposite side. For purposes of illustration, one might compare the arrangement with the connection of a telephonic apparatus with telegraphic wires, and the passage along them of vocal messages, for which originally they were not intended, and which, indeed, would never have been sent but for the breaking down of the wires by which they were normally transmitted. This may seem a somewhat extravagant theory, and it may be urged that no connection has been shown to exist between the decussating and non-decussating fibres. On the other hand, it seems exceedingly probable that in the intricate anastomosis of fibres in the cord, there may be some such connection, and the fact that sensory impulses can be conducted, although all motion is lost, when the cord is very considerably disorganised, suggests that when certain fibres are destroyed, their functions can be superadded to those still intact. A similar process must take place in aphasia, for it can hardly be imagined that the third right frontal convolution is a mass of non-functionating material, and yet it may undertake the lapsed duties of the corresponding convolution of the opposite side, and form endless connections with neighbouring centres, *e.g.*, the visual, auditory, arm, &c., in order to carry on the function of speech. It will be noticed in the cases quoted by Obersteiner that, in spite of the grave sensory disturbances, discrimination of heat from cold is still possible, and this function is probably conducted along certain of the non-decussating fibres of the antero-lateral tract, which must therefore remain uninjured. It will also be noted that, although a heavy touch is referred to the other side, a slight one remains unperceived, and is therefore non-conducted. This rather tends to suggest that there is a certain amount of resistance offered by the new path before it is completely opened out, and that therefore a considerable peripheral sensory excitation is necessary before it is able to overcome this resistance and be conducted to the brain.[1] Gowers mentions a patient, the subject of tabes, in whom a touch was referred to corresponding points on each side of the body at the same time. In this case I should imagine that the decussating fibres were in great part destroyed, but not wholly, and that common sensory impressions found their way up both these and the non-decussating fibres, on account of the relative insufficiency of the former. Such impressions would therefore be referred to both sides of the body.

To recapitulate. All the cases of allocheiria I have been able to find recorded, with the exception of that occurring in the course of hysteria, which sufficiently accounts for anything *bizarre* were accompanied with considerable ataxia and profound disturbance of common sensations. These symptoms are probably due to an affection of the postero-median columns, with the posterior root zones, and include the sensory decussating fibres. The sense of discrimination of heat from cold is probably conducted by non-decussating fibres in the antero-lateral touch. These are as yet unaffected, as in no case was the sense reported lost. The common sensory stimuli, finding their usual channel closed against them, are conducted upwards by the non-decussating fibres, and are consequently referred to the opposite side of the body. This will probably be regarded as very fanciful, and indeed, the theory is only suggested *faute de mieux.*

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[1] Diseases of the Nervous System, vol. i., p. 295.

THROMBOSIS OF INFERIOR CEREBELLAR ARTERY.

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Cases of thrombosis of the cerebellar arteries, causing acute softening, are sufficiently infrequent to merit individual mention.

Ann J., 27, married, was admitted on 17th May, 1890, suffering from early general paralysis.

State on admission.—Physical—Right pupil 5 mm., left 3.5, former inactive to light, latter hypersensitive, both accommodating to distance. The margins are slightly irregular and they are imperfectly circular. Optic discs and retinæ normal. Plantar reflex and patellar jerk dull, the usual facial and lingual tremors present, but not prominent. No ataxia or paralysis, no eruptions, cicatrices or nodes. Copious, dark-red, fœtid lochia, and other usual signs of recent confinement; temperature normal.

Mental.—There is some exaltation and loss of attention, but coherence, rationality, and recent and remote memory are normal. Gives this history: Has been married nine years to a disreputable husband. He deserted her some months ago, and she has since lived with a navvy on the Manchester Ship Canal. Has been somewhat intemperate herself. Pregnancies:—(1) miscarriage, (2) and (3) "7-month" still-born children, (4) and (5) "9-month" still-born children; the last confinement occurred ten days ago.

Progress of case.—Had some transient attacks of delirium during convalescence from confinement, but no rise of temperature. From 1st June till 1st November, 1890, her mental state may, in this connection, be considered normal, and she proved a steady and reliable worker.

Final illness: 1890, *Nov. 1st.*—Complains of severe general headache with gastric irritation, furred tongue, anorexia, no vomiting.

Nov. 2nd and 3rd.—No material change; is quite sensible.

Nov. 4th.—Vomited breakfast, took no more food during the day; has much nausea, headache continues severe. Was wakeful last night. Is weak and giddy, and sways on standing; no true ataxia. Reflexes all depressed.

Nov. 5th.—Does not attempt to stand, saying the head-pain is too severe. Localises it to occiput and forehead. Is dull mentally, but can be easily roused. Utters the "hydrocephalic" moan at intervals. At 4.30 p.m. suddenly jumped out of bed and rushed down the ward, crying out "Murder." Sank down in a heap at the door. The excitement passed in five minutes. Towards evening stupor became prominent.

Nov. 6th.—Lies huddled up in bed, uncomplaining and nearly unconscious. Cannot articulate answers to questions, but mental reflexes remain, and she protrudes her tongue on request. Pupils continue as before, 3.5 mm., nearly inactive to light; pulse 106.

Nov. 7th.—At 9 a.m. drooping of left eyelid was observed, at 11 left ala nasi, at mid-day left side of mouth, and tongue was protruded to left. During the afternoon the furrows of left side of face gradually disappeared. Pulse 115 per minute. Coma not yet absolute.

Nov. 8th.—Some diffuse weakness of left arm and leg. Left plantar reflex and patellar jerk are now in excess. There is slight left ankle clonus. Coma is absolute. Pupils and temperature as before.

Nov. 9th.—Increased flaccidity of left side. Left patellar jerk very active and ankle clonus brisk, right jerk exaggerated, no clonus. Left arch of diaphragm motionless, respiration spasmodic, no cyanosis. Temperature began to rise towards evening; at 7.30 was 99.6° F.

Nov. 10th.—General muscular flaccidity, all reflexes absent, respiration irregular, gasping and shallow; cyanosis and sweating present. Temperature at 7 a.m. 99° F., at mid-day 99.8°, at 4 p.m. 101.4°, at 8 p.m. 103.4°. Thence it fell until death at 10.30 p.m.

Post-mortem 13 hours after death.—Moderate rigidity. We may summarise that the brain was firm, little wasted, and showed the usual signs of early general paralysis, that the vessels were widely affected with endarteritis, but showed no atheroma, and that other syphilitic lesions were absent. The outer and posterior aspect of the inferior surface of the left lobe of cerebellum was extensively softened and pulpy, and the condition extended at least 2 cm. into the substance of the organ. A like condition was present in the left inferior cerebellar peduncle, and extended down the cord to the middle of cervical enlargement, gradually lessening in degree and extent. On the left inferior cerebellar artery, 1 cm. from its origin from the vertebral, was a sclerosed patch 4 mm. × 2 mm. Beyond this the vessel was empty, but a clot spread from it to the vertebral, and thence down the latter vessel as far as the foramen magnum, entirely blocking the lumen. At first it was firm and partly decolorised, but lower down quite recent. The vertebral above the origin of the inferior cerebellar was free and empty. The left arch of diaphragm reached to the upper border of the fourth rib, two spaces higher than usually found. The right was in the ordinary position, reaching to the upper border of fifth.

Remarks.—This case is incomplete, because the cerebellum, medulla and cord, with the vessels, which had been set aside to harden, were accidentally thrown away, before sections were cut. No opinion, therefore, can be expressed as to the limits of the lesion, the centres affected, or the presence of other vascular obstructions. But the clinical aspect remains, and possesses a certain negative interest in supporting the ordinarily accepted theory that lesions of the lateral lobes of cerebellum produce, *per se*, no evident diagnostic symptoms. We may assume, from the patient's own account of her pregnancies and habits of life, and the evidence on admission of her having lately given birth to a dead fœtus (copious putrid discharge without septic absorption), even in the absence of an independent history, that she was syphilitic ; and shall probably be correct in referring the arteritis and general paralysis to this cause. But further deduction is unsafe. Thus, the headache was at first supposed to be functional, due to over-eating ; and when, three days later, its organic nature seemed likely, the mental torpor was already too pronounced to permit of an exhaustive search for psychical changes of any kind. The pupils and other reflexes were put out of court from the first, owing to the presence of general paralysis. It is probable that the localising signs which appeared on the sixth day of illness denoted the advance of the clotting process from the inferior cerebellar along the vertebral, interfering with the nutrition of the medulla, and that the fatal event was retarded for three days owing to the free anastomoses chiefly through the anterior spinal. It will be noted that the 7th and 12th nerves were earliest affected, and that evidence regarding the 8th, 9th, 10th, and 11th, is indeterminate. The diagnosis was arachnoid hæmorrhage (the so-called "pachymeningitis hæmorrhagica interna") on the inferior surface of cerebellum, spreading down the side of the medulla, and exerting pressure on the nerve roots, and later, on the columns. Probably the extreme rarity of that lesion in this situation should have prompted a more correct guess.

POLYURIA (DIABETES INSIPIDUS) IN BASAL CEREBRAL DISEASE.

BY HENRY HANDFORD, M.D., M.R.C.P.LOND.
Physician to the General Hospital, Nottingham.

THE following two cases are the only examples of this phenomenon with which I have met in a somewhat extensive hospital experience. And the second, in which the fact rests only upon the statements made by the patient and her friends, is sufficiently indefinite to be cast aside, were it not so amply supported by the details of the first case.

Gowers says, "The urine is seldom altered in composition by organic brain disease, although polyuria rarely, and glycosuria occasionally, have been produced by disease of the pons and medulla oblongata."

The analogy with diabetes from puncture of the floor of the fourth ventricle seems to be very close. It would appear probable that the pressure of a gumma paralysed the vasomotor centre for the kidney in the medulla, the absence of polyuria until the latter part of the second attack, and its continuance when all other symptoms were improving, is difficult to explain. But we must accept as a fact that a tumour may grow all round a nerve without pressing upon it sufficiently to interfere with its function, and that of several nerves to all outward appearance equally involved and pressed upon by a tumour some may be completely paralysed, and others quite uninjured. The direct and indirect pressure upon the brain substance is also open to many sources of variation. Hence it is useless to speculate upon the escape of the renal centre until a particular time, and the absence, or slight degree of, improvement when the other symptoms of pressure upon the nerve trunks and nerve tissues were so greatly diminishing.

The frequency with which iodide of potassium and mercury are given for diseases of all kinds without any such diuresis following, renders it clear that the treatment was not in any way responsible for the phenomenon.

I have detailed the first case very fully in order that it may bear internal evidence of the accuracy of the diagnosis, both as to the presence of a tumour and as to its syphilitic nature.

Case 1.—Julia M., aged 27, lacewinder, admitted into the General Hospital, Nottingham, Oct. 15, 1891, complaining of fits, &c.

Her father died in the hospital of chronic Bright's disease. Her mother died of dropsy: brothers and sisters are healthy. Patient had fits as a child, and five years ago fell and struck her head, remaining unconscious for two or three hours. She completely recovered. Before marriage she was for some years a prostitute; had venereal disease, rashes, and sore throat. She has been married 6½ years, and has two children, the youngest eight months old.

Her present illness commenced about eight months ago, three weeks after her last confinement. She noticed that her left eye was getting "crooked," and she frequently vomited. (The vomiting continued till six weeks ago).

She next found that her left arm and leg were getting weak. She could not hold the baby on her arm.

She suffered from double vision, headache, and a feeling of pain, and "pins and needles" shooting down the left side. This peculiar numb feeling usually only continues about five minutes, but when it lasts a quarter of an hour or longer she loses consciousness. She can tell when these "fits" are coming on.

On admission she was found to be well nourished and fairly intelligent, but her memory was evidently failing.

The grasp of the left hand is markedly weaker than the right:—dynamometer, L. 35, R. 65. The left leg is also evidently weaker than the right. There is no facial paralysis, and the tongue is protruded straight. Sensibility to touch and to pain are normal, except perhaps over the left arm, where they are both somewhat deficient. The muscular sense is impaired on the left side. Sensation to heat and cold normal. Taste impaired, but not lost.

The plantar reflexes are exaggerated, especially on the left side; the patella reflexes also; no ankle clonus. The left wrist, triceps and biceps jerks are exaggerated.

There is complete paralysis of the right external rectus; and marked weakness of the left external and internal recti. There is nystagmus on the left side, and some proptosis. No optic neuritis, but masses of pigment showing remains of old choroiditis. Left eye myopic, right hypermetrophic.

Ten days after admission the note runs:—Since admission she has had 3 to 9 fits daily, never losing consciousness. She has vomited once or twice for the past two days. The headache has been more severe, and is general in distribution. It is better at night and allows her to sleep. She is up in the afternoon and can get about.

Sensory Aura.—She always knows when the fits are coming on by a pain in the left temple, followed by a feeling of "pins and needles" in the left arm, commencing in the fingers and passing up the arm. This is from five to ten minutes before the fit commences. After the "pins and needles" in the arm comes a pain in the left flank just below the tenth rib, passing down the outer side of the thigh and leg to the toes which are then flexed, and immediately the leg becomes rigid and remains extended. She then rubs the left foot with the right. In the meantime the fingers of the left hand are flexed, and the arm becomes extended until she grasps the wrist with the right hand and draws it across the chest. She does not utter any cry. The fit lasts about two minutes. The mouth is drawn a little to the left. The procedure is exactly repeated every time. She becomes drowsy after the fits, and complains of the headache becoming much more severe and general.

Nov. 2.—She has been taking iodide of potassium, gr. x., three times a day, and pil. hydrargyri gr. 3¾, twice a day, ever since Oct. 20. She is now gaining power in the left arm and leg, and the fits are not nearly so frequent. The aura has ceased, so that now the fits come on without warning. And instead of being preceded by tingling in the leg, they are *followed* by pain in the left middle finger going up to the elbow. The pain in the head has been much less severe.

Dec. 2.—For the past few weeks she has never had more than one slight fit daily and often none for several days.

Now the fits *commence* in the leg with rigidity of the muscles; the tingling and sensory phenomena have ceased.

Jan. 8.—She has been away to the convalescent hospital for a fortnight, and during that time has only had one fit.

The left leg seems now as strong as the right, and she can walk several miles. There is no stiffness, but the left knee-jerk is greatly increased.

She has had no headache for several weeks. The reactions of the pupils are normal, but the left is slightly larger than the right. There is still weakness of both external recti and nystagmus on abduction. No change in the fundus besides the remains of old choroiditis. She is now an out-patient.

Jan. 21.—Since she left the hospital a perforation of the soft palate has formed close to the uvula, which seems likely to slough off. There is also a syphilitic pharyngitis.

March 7.—Patient was readmitted. She had not attended since Jan. 21.

She has had much headache and vomiting; and is now very feeble and emaciated. She is hardly able to stand, and usually lies in a semi-comatose state. The left knee jerk is exaggerated, the right almost absent. There is no distinct clonus on either side, but some slight rigidity of both legs. Slight impairment of common sensation over the right hip and flank. The condition of the palate and fauces is unaltered since Jan. 21st. She can move the tongue freely: it is dry and coated with a brown fur. Temperature 97.2.

There is complete paralysis of the left external rectus, and nearly complete of the right, Pupils more than half dilated. Left disc swollen: vessels curving over the edge. Right disc slightly hazy. Left globe prominent.

April 4.—Patient has been treated by iodide of potassium, gr. x, three times a day, together with mercurial inunction.

For a few days after re-admission she was delirious, especially at night; and for a week or two she lay in a semi-conscious, drowsy, state; for the first fortnight the urine was scanty.

From *March* 15 the quantity of urine almost suddenly began to increase enormously, ranging from 100 oz. to 130 oz. in addition to what was lost with the motions. The sp. gr. varied between 1,006 and 1,008; and the urine was quite free from albumin and sugar.

From *April* 12 to May 2 patient again went to the convalescent hospital and improved in general condition, but the urine still continued between 100 and 140 ounces daily, besides what was necessarily lost.

On *May* 25 the iodide of potassium was stopped, and ℥i. of liq. hydrargyri perchloridi alone given. The quantity of urine was not materially affected, although it continued to be carefully measured for ten days further. She was then made an out-patient.

A month later, June 22, she was again re-admitted for two days, and the polyuria still continued. The paralysis of the external recti, the perforation of the soft palate, and the exaggerated knee jerk remained. Otherwise she seemed well. She was advised to continue medicine once a day for another six months; but she did not return, and has been lost sight of.

The second case is not nearly so definite. She was admitted in January, 1890, under Dr. Ransom, to whom I am indebted for permission to use the case.

The patient was a woman, aged 34, who had had two children and three miscarriages, with a very definite history of syphilis.

During the past year she has suffered much from headache. In April, 1889, *i.e.*, nine months ago, she began to suffer from intense thirst. In one afternoon she drank two dozen half-pint bottles of soda water. She did not drink intoxicating liquors. In one day she has passed as much as three gallons and half a pint of urine.

This polyuria ceased two months ago, when the headache became more severe, and drooping of the left eye-lid began. She has had no vomiting.

She is slow in answering questions, and seems to forget words. There is no paralysis of face, arms, or legs; but there is complete ptosis of the left eye-lid, and the left pupil is dilated and does not re-act to light or accommodation. No optic neuritis. Sensation is unaltered. She has had several slight epileptic seizures.

She was treated with pot. iod. gr. x., three times a day, and improved considerably. The polyuria did not return while she was in the hospital. A gumma was diagnosed in the temporo-sphenoidal lobe, involving the third nerve as it emerges from the base of the brain.

A CASE OF FRACTURE—DISLOCATION OF SPINAL COLUMN, WITH LESION OF THE CORD.

BY F. W. TUNNICLIFFE, M.B.LOND.
Late House Surgeon, St. Bartholomew's Hospital.

FRACTURES and fracture dislocations of the spinal column with consequent lesion of the cord being of fairly common occurrence, perhaps some excuse will be expected for publishing this one. The chief interest of the case, I think, lies rather in the completeness and suggestiveness of the symptoms and in the microscopic morbid anatomy than in the trauma, and this is my reason for wishing to publish the case; for permission to do this, as well as for supervision throughout, I am indebted to Mr. Bruce Clarke.

J. M., aged 36 years, a builder, was admitted into Darker Ward, under the care of Mr. Bruce Clarke, on Jan. 27, 1892, suffering from a fracture dislocation of the spinal column.

History of present condition was as follows:—

On the afternoon of admission patient fell 30 feet from the top of a ladder, and when on the ground the ladder fell on his back.

Family history and past history both irrelevant.

Patient is a big heavily-made man, height 6ft. 2in.; upon admission, temperature 98° F., pulse 80; evident signs of dyspnœa.

Upon examination a lump was found in mid-dorsal region, with a hollow immediately above, this projection was apparently formed by the 6th dorsal vertebra, and its prominence was due to the fact that the column above was displaced forwards.

The following marked functional disturbances were present:—

A. Below line of lesion.

(1) *Sensation.*

Complete loss, including loss of sensations of—

(*a*) Touch.

(β) Pain.
(γ) Heat and cold.

(2) *Motion.*
(a) Complete paralysis, respiration entirely diaphragmatic.
(β) Idiomuscular twitchings of great pectoral muscle.
(γ) Retention of urine.

(3) *Trophic.*
Factitious urticaria, tâche cérébrale could be produced anywhere below level of lesion.

(4) *Sweating.*
Sensible perspiration absent below lesion.

(5) *Reflexes.*
(a) Cutaneous reflexes, plantar and cremasteric, *present.*
(β) Deep reflexes, patella, ankle clonus, *absent.*

B. Above line of lesion.

(1) *Sensation.*
(a) A narrow zone of imperfect sensation, tactile, pain, heat and cold feeling being equally diminished.
(β) A zone, about 3 inches wide, of marked hyperæsthesia, the slightest touch causing pain.
Application of a test tube containing water at about 90° F., sensation of scalding.
(2) Motion normal.
(3) Sweating profusely.
(4) Deep and cutaneous reflexes normal.

Vide Diagram.

Urine drawn off with a catheter (no uretheal spasm) acid 1025, no albumen, no sugar.

Temperature upon admission normal. Pulse and respiration also normal.

In addition to the spinal column injury, there existed also a fracture dislocation of the manubrium sterni.

The manubrium, with the two first ribs, being displaced backwards and downwards. No fracture of the ribs was made out.

The treatment consisted in making extension, under chloroform, on the shoulders and head, while the pelvis and the feet were fixed, with also at the same time manipulation at the seat of fracture: this succeeded in reducing partially, but not entirely, the dislocation; the extension and counter extension were performed with the patient on his side, he was then rolled over on his back on a water bed. His head was kept in a horizontal position, and was prevented from moving from side to side by sandbags.

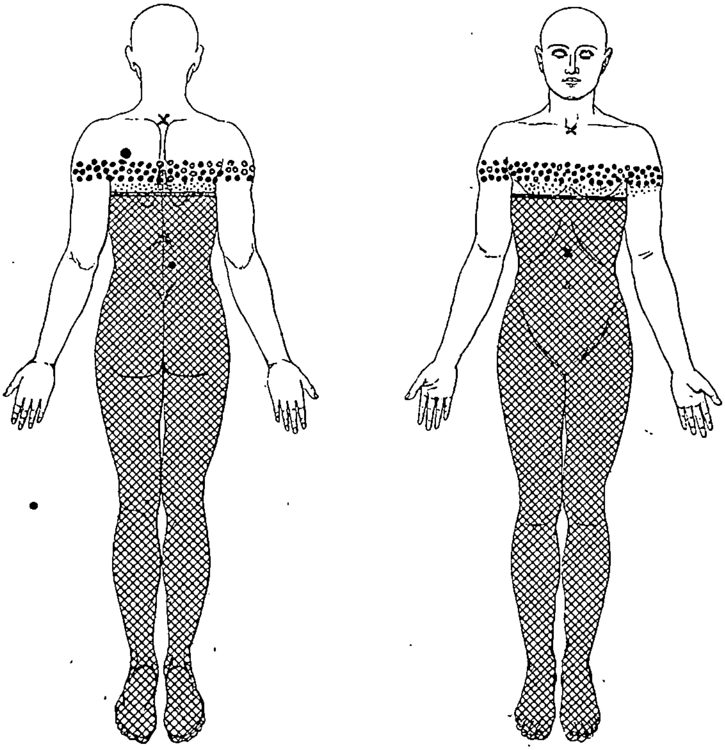

DIAGRAM I.

——— Line of lesion.
× × 7th cervical and 1st lumbar vertebræ.
∴∵∴ Zone of hyperæsthesia.
⁞⁞⁞⁞ Zone of imperfect sensation.
xxxxxx Region of complete anæsthesia and paralysis.

Jan. 28.—Patient passed a very restless night. Morphine gr. $\frac{1}{4}$ administered. Respirations rapid (32 per minute) and laboured.

Temperature 99° in axilla. Patient still unable to pass urine. Catheter passed every 6 hours.

Jan. 29.—Dyspnœa more marked. Resp. 40. Patient is becoming cyanosed. Pulse 120. Temperature 10° F.

Patient continued to get worse rapidly. Death occurred 2 a.m. Temperature at time of death 106.6° in rectum, 102·6° in axillæ, 102.8° in mouth.

The *post-mortem* examination made the day of death, Jan. 30, revealed the following conditions :—

Both pleuræ contained large quantities of blood. Contents of pericardium normal. No rupture of large thoracic vessels made out, hæmorrhage probably arose from rupture of internal mammary veins which was dependent upon the fracture dislocation of the manubrium sterni.

An oblique fracture was found through the manubrium sterni; direction of fracture was from above, downwards and backwards. The upper piece was driven backwards and downwards, and the two first ribs with it.

Spine.—The laminæ of the 4th, 5th, and 6th dorsal vertebræ were fractured, on opening canal there was some blood outside theca, but not enough to cause pressure on the cord, within the theca there were no clots visible. The cord was entire as regards its continuity; at the level of the 6th dorsal vertebræ cord was swollen and bluish. The bodies of the 4th, 5th, 6th dorsal vertebræ were fractured.

Microscopical Examination of the Spinal Cord.—The portion of the cord corresponding to the seat of injury was hardened in Müller's fluid, embedded in celloidin, and the sections were stained in logwood and eosin. Portions were also excised from the cervical, and lumbar regions of the cord, and were examined in the same way.

A few sections from the centre of what appeared to be the most bruised part were chosen for microscopical examination, and then a few sections from the upper and lower limit of this until at either extremity healthy cord was reached. The effect of staining with logwood and eosin, for which suggestion I am indebted to Mr. Austen, is to stain all hæmorrhagic patches dull red, while the rest of the cord is colored bright purple. The sections upon microscopic examination shewed that the cord had been crushed. The central canal was no longer recognizable.

The entire grey substance was the seat of scattered patches of hæmorrhage, the hæmorrhage extending along the anterior and posterior nerve roots. Of the white matter, the posterior and postero-lateral columns were most affected.

Upon the use of a higher power the individual eosin stained blood corpuscules composing the dull red patches could be distinctly recognized. Sections above and below this, the most injured level, shewed a gradual diminution in extent of the hæmorrhages, this could be traced section by section until healthy cord was reached.

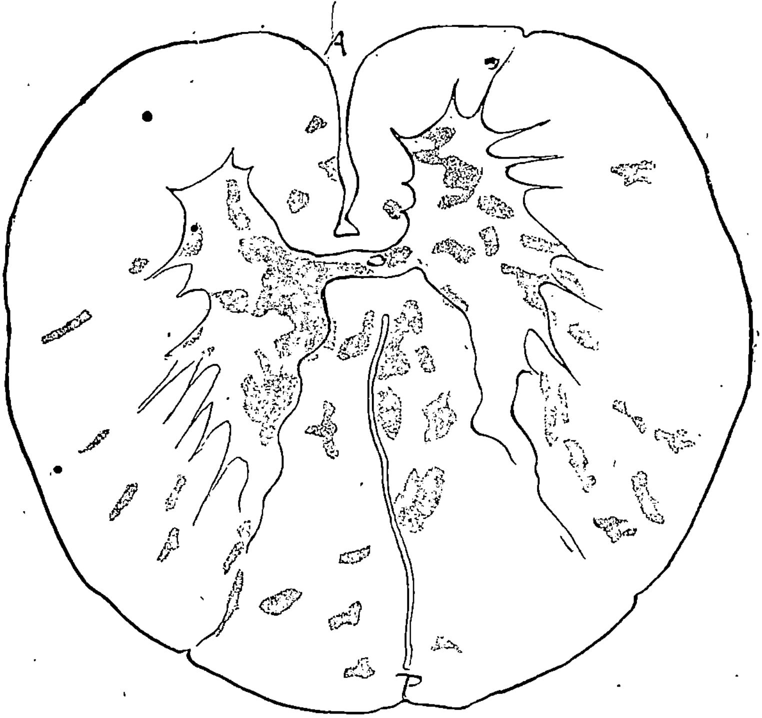

DIAGRAM II.
A anterior, P posterior, longitudinal fissure of the cord.

The result of the microscopical examination of the lumbar cord at the level of the lumbar enlargement was to confirm the macroscopic appearance, viz., to show nothing abnormal.

The adjoining diagram illustrates a section, taken from the centre of the bruised portion of the cord. The shaded areas mark the positions and extents of the hæmorrhages. For this diagram, as for the preceding ones, I have to thank Dr. Adamson.

Remarks.—There is, I venture to think, considerable interest in this case on account of, at any rate, two phenomena. I refer to the absence of sweating below the lesion and the high temperature in the rectum, as compared with the axillæ and mouth. I will not venture to discuss a phenomenon which is also interesting, viz., the complete abolition of the deep reflexes below the lesion, this I am aware can be attributed to shock. I will merely remark, *en passant*, that the cutaneous reflexes were present, also that the so-called "shock" was accompanied by stimulation of the vaso-constrictors, as evidenced by the dryness and pallor of the skin below the lesion. The idea of nervous shock, although the turn is as indefinite as it can be, is usually associated with that of paralysis, of dilation of the vessels of the skin, in short with sweating; here at any rate this was not the case, there was moreover, no evidence of paralysis of the sphincter ani or vesicæ, in other words there was no evidence of "shock" below the lesion other than the abolition of deep reflexes.

It is also to be noted, in view of recent[1] theories upon this subject, that no myelitis existed elsewhere, more particularly that the lumbar enlargement was healthy.

With regard to the profuse sweating above and the entire absence of sweating below the lesion, I know of nothing in any way similar.

It must also be remarked that the skin was very much redder above than below the line of lesion; in other words, there was dilation of dermal vessels. This profuse sweating, which was much more marked during sleep, cannot be attributed to the temperature, this being only 99° in axilla and mouth. Dr. Brunton[2] has sought to attribute the night sweats in phthisis to the increased amount of carbon dioxide in the blood, which itself is due to the diminished respiratory capacity of the individual. Leaving out of consideration why they occur at night, the fact remains that the respiratory capacity of the patient in question was suddenly considerably diminished, by the occurrence of a double traumatic hæmatothorax. This taken in connection with the fact that the patient's sweating area was lessened would probably explain the diaphoresis above the line of the lesion.

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[1] Gowers. Spinal Cord, 2nd edition. Lond., 1892.

[2] *St. Bart.'s Hosp. Reports*, vol. xv., "Pathology of Night Sweating in Phthisis."

The most interesting phenomenon remains, viz., the complete absence of sensible perspiration below the line of the lesion, this being undoubtedly accompanied by constriction of the vessels of the skin, as evidenced by the surface temperature (a surface thermometer was not used) and the paleness of the skin. The question naturally arises, was the absence of sweating entirely due to the diminished blood supply, in other words, was it merely the expression of a vaso-motor change, or must we look to some concomitant change in the origin or centres in the cord of the secretory nerves distributed to the sweat glands. We know perfectly well that skin pallor can occur with diaphoresis on the one hand, as in fright, nicotine poisoning, &c., and also skin redness with complete absence of sweating, as in atropine poisoning, on the other, thus shewing distinctly that the two mechanisms, although generally acting together, can each when occasion requires, act separately. We must, I think, conclude that both causes were here at work to cause absence of sweating.

With regard to the temperature, unfortunately the only comparative observation was taken just before death, this shewing a difference of 3.8° between rectal and mouth temperature at the same time. Whatever this may mean, one cannot help noticing that the high temperature was like the absence of sweating, below the lesion, in other words, that relatively to the lesion in the cord, high temperature was associated with diminished heat loss.

A CASE OF INJURY TO THE MEDIAN NERVE ILLUSTRATING THE POWER OF REPAIR OF A MIXED NERVE TRUNK; AND THE MODE OF ACTION OF PILOCARPIN.

BY THOMAS D. SAVILL, M.D.

In Infirmary practice one often has the opportunity of observing a patient over a long period of time, and the following case, which has been under my notice for nearly six years, may be interesting.

It illustrates the power of repair of a mixed nerve trunk, where the injury is of a very definite and known extent.

Daniel Flynn, who was then 20 years of age, a bargeman, was admitted to the Paddington Infirmary on September 20th, 1887, for tetanus. The case was one of extreme severity, and as there was a clear history, 14 days before admission, of injury to the palmar twigs of the median nerve by falling on a dust heap, it was thought that division of the nerve might possibly give him an additional chance of life.

Accordingly upon *September 28th*, 1887, *half-an-inch of the median nerve trunk in the upper arm was removed, the two ends being loosely connected by cat-gut, leaving $\frac{3}{8}$ of an inch interval.* The wound healed by first intention; and this operation was, of course, followed by paralysis of the muscles, and anæsthesia of the skin supplied by the median nerve. These symptoms, it was hoped, would pass off after the nerve had re-united. But it was not so; the weakness of the muscles increased, they became atrophied, and presented marked reaction of degeneration.

At the end of three months (*Dec.*, 1887) blebs formed spontaneously on the knuckles of index and second fingers of the right hand. These blebs soon became open sores (and did not heal for any great length of time during the next 13 months, fig. 3). About this time, also, it was noticed that the skin over the outer half of the hand was rougher and redder than that over the inner, and that this half of the hand perspired a good deal more than the other, pointing to a vaso-motor lesion. He was treated with galvanism, massage and faradism without result. The accompanying sketches made for me by Dr. G. W. Davis, at the bedside,

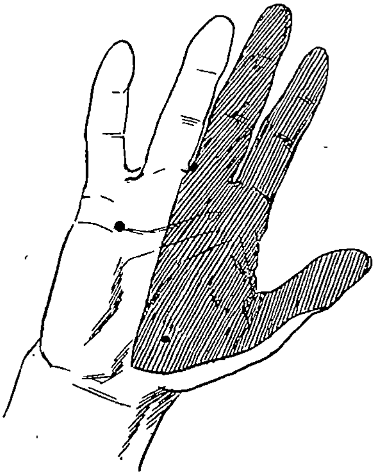

FIG. 1.

Shows area of complete anæsthesia, December 22nd, 1888.

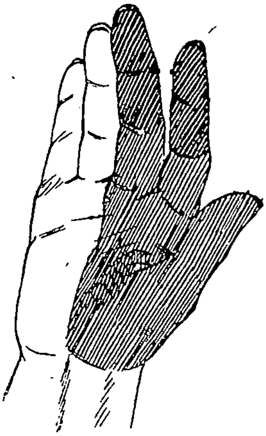

FIG. 2.

Shows area of complete and partial anæsthesia, May 7th, 1888.

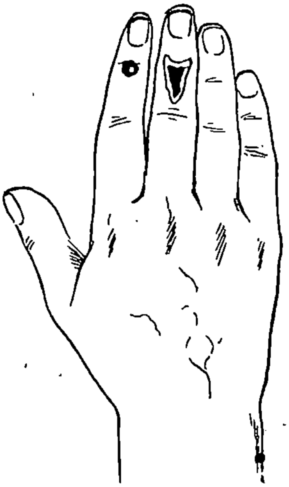

FIG. 3.

Sores on knuckles, May 7th, 1888.

show the state of the anæsthesia at that time. It will be noticed that in May, 1888, the area of complete anæsthesia extended only just above the first phalangeal joint.

In *June,* 1888, eight months after the original section the continuity of the median nerve had been restored to some extent, for slight power of movement had returned in the fingers, and acute pain was felt on pressing over the nerve trunk in the fore arm and in the upper arm below the injury. But the nerve was obviously the seat of some irritation, and I therefore decided to cut down and explore the nerve trunk at the seat of the original operation, where a swelling in the nerve could be felt.

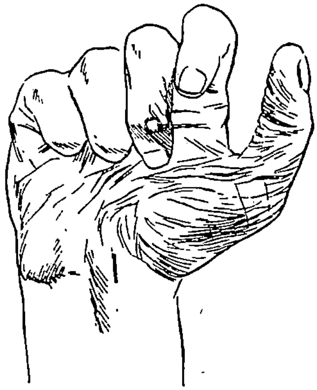

FIG. 4.

Shows maximum closure of fingers and thumb, September, 1888, one year after the first, and three months after the second operation (resection). Also shows atrophy of muscles of thenar, wrinkled skin over outer half, and position of bleb on knuckle. The muscles of the forearm were also much atrophied.

On *June 8th*, 1888, one inch and a half of the median nerve trunk including a fusiform fibro-neuroma was removed, and the healthy cut ends brought together by two cat-gut sutures as closely as it was possible; that is to say, within half-an-inch of each other. The wound healed by first intention. The limb was put up in plaster with the forearm semi-flexed, and kept so for ten weeks.

For several months after the operation the patient suffered a great deal of pain down the median nerve, which was so much increased by extending the arm that it had to be kept flexed. Three months after the resection (*September*, 1888) motor power began to return. The accompanying sketch (fig. 4), from a photograph made at that time shows the maximum power of closure of the fingers and thumb, and other particulars, three months after the second operation. There was considerable atrophy at this time, not only of the flexors of the forearm and the thenar muscles, but also of the bones, for there was a shortening and thinning of the fingers; the photo also shows the wrinkled state of skin on the outer side of the palm. Fresh sores formed, this time on the tips of first and second fingers. Sensation had not returned; it was now the same as shown in fig. 2.

With a view of ascertaining whether pilocarpin really excites sweat through the sweat nerves, the following experiment was performed on *Nov. 9th*, 1888.

"11.10 a.m.—Condition of hand as usual—inner side sweating, outer side slight blush. Pilocarpinæ nitratis gr. $\frac{1}{10}$ injected into *left* arm. In five minutes the outer side became more red than inner side, but no alteration of perspiration. In ten minutes no further effect, so $\frac{1}{10}$ gr. more injected.

"In fifteen minutes outer half of palm (*i.e.*, part supplied by median nerve) very red, much redder than inner side.

"Inner side perspiring profusely, outer side not at all.

"Profuse perspiration all over body."

In *February*, 1889, eight months after the resection he left the Infirmary. At this time power had, to some extent, returned in the muscles (dynamometer—right 39, left 78), and he could close the fist completely, though the movements were stiff. There was pain experienced at the seat of operation, stiffness on extending the forearm, and tenderness down the median nerve. The sores on the fingers had quite healed now. The atrophy, condition of skin, and anæsthesia all remained the same.

June, 1889.—He went to a convalescent home for some months, and on his return to the infirmary in *June*, 1889 (one year after the resection), power of motion had still further improved (dynamometer—right 51, left 85). About this time sensation began to improve; but atrophy still remained the same.

In the *Autumn of* 1889 (two years after the original operation and one-and-a-quarter after the re-section), the muscular atrophy began to improve, and in March, 1890, there was only ½ inch difference in the measurements of the two forearms.

At the present time, *June*, 1893 (five years after the resection), there is still some deficiency of power (dynamometer—right 52, left 86), and also slight stiffness; but he has been able to follow the occupation of omnibus conductor during the last three years with perfect success, excepting that he has to use his left hand to sort the money in his bag on account of stiffness of the first and second fingers of his right hand. There is no anæsthesia or analgesia now; he distinguishes heat and cold, and his muscular sense is good. The skin of the outer half of right hand is now paler than the corresponding part of left, there is still some tenderness over the course of the median nerve in the forearm, and still scars from the old blebs on knuckles and tips of fingers.

As regards nutrition of muscles, the two forearms now measure precisely the same, and the two thenar eminences are the same, showing that the muscles have fully regained their volume. But it is not so as regards the bones; there is ¼ inch difference in the circumference of the two wrist joints; and the right thumb, first and second fingers are measurably shorter and thinner than those of the other hand.

The following measurements are in centimetres:—

RIGHT.		LEFT.	
Wrist	15.8	Wrist	16.5
Round distal joint of thumb	6.1	Round distal joint of thumb	6.5
,, ,, ,, index finger	4.6	,, ,, ,, index finger	5.2
,, ,, ,, second ,,	4.8	,, ,, ,, second ,,	5.2
Total length of thumb......	6.5	Total length of thumb......	7.0
,, ,, ,, index finger	9.5	,, ,, ,, index finger	9.5
,, ,, ,, second ,,	10.8	,, ,, ,, second ,,	10.8
Round hand opposite cleft of thumb	20.6	Round hand opposite cleft of thumb	22.4

Remarks.—The injury at the time of resection (in June, 1888) was very considerable, and was of well-known extent; 1½ inch was removed. The nerve trunk on the peripheral side was already in a state of degeneration (as shown by the microscope). The ends could not by employment of any reasonable force be brought together closer than ½ an inch.

The chief points of interest are:—

(1) The extent of recovery in the muscles. They have now (June, 1893) quite resumed their former volume, and very nearly their old strength. The patient only complains of a little stiffness in the fingers.

(2) The permanent atrophy of the bones.

(3) The complete restoration of sensation.

(4) The pilocarpin experiment, producing extreme redness over the palmar branches of the median; but no perspiration, such as elsewhere occurred, over this part of

the hand. It shows that this drug acts through nerves conveyed in the median trunk.

His remarkable recovery from tetanus, under treatment by large doses of chloral hydrate, was the subject of comment elsewhere.[1]

The recovery of the nerve may, to a large extent, I believe, be attributed to the limb having been kept at perfect rest.

I was inclined at one time to take a very gloomy view of the prognosis, and contemplated the transplantation of nerve from an animal, as in Gersung's case.[2]

But on reading in Hilton's "Rest and Pain" of three cases of injury to nerve which recovered after several years' rest, I prescribed this treatment for my patient, and the result has been most satisfactory.

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[1] *Lancet*, November 24, 1888.

[2] *Brit. Med. Journal*, May 19, 1888.

Abstracts of British and Foreign Journals.

Model of the upper part of the Spinal Cord and the Oblongata.[1]

By Dr. Ludwig Edinger, Frankfort-on-the-Maine.

The knowledge of the inner structure of the central nervous system is generally acquired by examining and combining transverse sections. As the delineations of these impress themselves especially on the minds of students, I have found it advisable, in my new model, to represent the fibres as issuing from the reproduction of a transverse cut.

A revolving pillar rises from a heavy pedestal; to the top of this is attached a horizontal bar—75 cm. long—representing the central canal. To one end of the bar is fastened a perforated metal plate on which the rhomboidal area of the medulla is painted, whereas the other end of the bar terminates in a similar reproduction of the cervical part of the spinal cord. The various nuclei and fibres are distinguished from one another by different colours. The proportions as well as the colouring have been so selected that minute details can be seen at a considerable distance.

The most important fibres and cells have been reproduced by means of wires and wooden models. Between the cross sections a number of variously painted wires are extended which represent connections of identical areas or continuation of the entering root fibres.

The cells of the spinal ganglia and of the motor and sensory nuclei are shown in their various forms. Larger aggregations of cells, of which it seems difficult to give a proper imitation in a model, as for example the forms of the nuclei of the posterior columns and the ascending gelatinous column of the trigeminus and glossopharyngeus are simply given in carved wood. In order not to make the matter too complicated the nuclei of the hypoglossal and vagus are given in the same form.

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[1] Author's translation from his paper in *Anatomischer Anzeiger*, 1893, No. 5.

As the model is designed to give a first insight into the subject, I have endeavoured to make it as simple as possible, and several tracts consisting only of a few fibres have been omitted. The name of each part is signified by a printed label. (In the wood cut, which illustrates this description, numbers are substituted.)

As few colours as possible have intentionally been used, and each tract retains its colour throughout. All motor root tracts are painted light red, the primary sensory tracts light blue. The secondary motor tracts are dark red, the central sensory tracts dark blue.

All the wires representing tracts which extend either caudad or frontad beyond the sections project at either end.

The model was completed in the spring of 1892, and shows matters as they were treated in the third edition of my "Vorlesungen ueber den Bau der nervösen Centralorgane," Leipzig, C. W. Vogel.

In studying the model it is the best plan to begin with an anterior root (light red wire, No. 32 in the cut). Motor roots enter into the transverse sections as well as into the intermediate part of the model, but only the cross section gives a complete view of the different ways they take. Every root fibre terminates in a multipolar ganglion cell. Round this cell twines a branch of fine red wires, the extremity of the secondary motor tract (red wires) which springs as a collateral either from the lateral (No. 26) or anterior (27) pyramidal tract. It is easy, in this manner, to follow these pyramidal fibres as far as the base of the medulla where the lateral tract decussates and both together form the ventral pyramidal area (No. 19) of the oblongata.

Of the sensory nerve (light blue, No. 30) a short peripheric portion is given, the greater part of whose fibres terminate in cells of the spinal ganglion (31). From these cells the roots enter the model and divide in the well known manner into different branches. Each part, however, is here represented by a single fibre only. The division of entering roots into ascending and descending branches is shown. One part of the posterior roots enters the posterior tracts (20, 21) in which it ascends and can be traced as far as the posterior nucleus funiculatus of the medulla. Another reaches the grey matter through the posterior grey column where its fibres either spread themselves around the cells or proceed to the Clarke's column. One fibre can be traced up to a cell of the anterior grey column. The direct tracts leading to the limiting layer of the grey substance as well as the fibres extending to the crossed posterior tract, have been omitted; likewise the posterior commissure, because our knowledge of the structure of the latter is too imperfect.

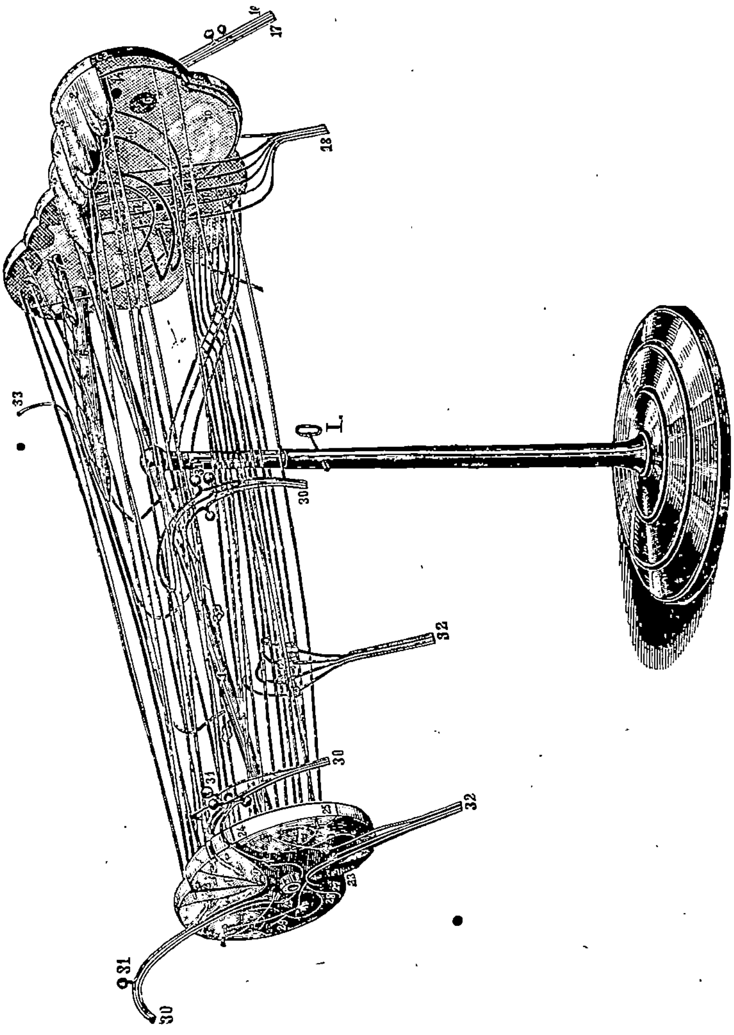
From the fusiform cells (dark blue) of the posterior column springs the secondary sensory tract (dark blue). It decussates through the anterior commissure into the antero-lateral tract, and may be followed up to the fillet (11). The (yellow) fibres of the lateral cerebellar tract spring from the cells of Clarke's column. 25 shows the position of the fasciculus antero-lateralis at the central margin of the above-mentioned lateral cerebellar tract. Lastly, in the spinal cord a single commissural cell is shown which sends its dividing fibre to the antero-lateral tract of the opposite side.

The secondary tracts which spring from the nucleus of the funiculus cuneatus and funiculus gracilis in the medulla are the following: On the mesial side the internal arciform fibres (15) crossing to the fillet, and laterally the external arciform fibres anterior and posterior (13, 14) to the restiform body (8, 9). So, by following the blue fibres it is easy to demonstrate the rather complicated course of the sensory tract as it is thought to be at the present time. Thus it strikes the eye that in the medulla all fibres are situated on the side opposite to that which they have entered in the root. Laterally from the oblongata cross section the accessory nerve (33), which may be followed to its knee, juts out. Of other cranial nerves, the ascending part of the trigeminus (6) and of the glossopharyngeus (7) is shown: their fibres, dividing into fine brushes, end in the gelatinous columns, which in our model are represented in carved wood. Besides, the hypoglossal nerve (18) and part of the vagus roots (16, 17) are represented; from the nuclei of the latter the secondary (central) fibres are seen crossing over to the fillet. Of the connection between medulla and cerebellum only the spinal part of the restiform body could be shown, because the fibres descending from the cerebellum to the olivary body (10) only appear in more central parts of the medulla.

N.B.—As only part of the fibres have been embodied in our model, a few hanging wires had to be supported by metal props. These props, as all other parts of the model which serve merely technical purposes, have been painted in black, in order to be distinguished from the essential wires.

Both cross-sections bear handles which are to be used for turning the model, in order not to dirty the bright colours by touching.

The model is manufactured in the laboratory of Mr. R. Jung at Heidelberg. It is durable, and solidly as well as neatly made; with the exception of the nuclei and gelatinous substance, it is constructed entirely in metal. Care has been taken, in designing it, that new wires may easily be inserted, as well as other changes made.



Diagnosis of the Diseases which Clinically resemble Tabes Dorsalis (Pseudo-Tabes, Neuro-Tabes, &c.).

By Dr. PAUL BLOCQ. *Gaz. des Hôpitaux*, March 22, 1890.

To simplify the differential diagnosis of the above affections a preliminary classification is adopted according to the presence of ataxy in

(1) Spinal affections; (2) diseases of the peripheral nerves; (3) neuroses.

In the following table the diseases in which tabetic symptoms are found are arranged in convenient order.

- *A*. Diseases in which tabetic (ataxic) symptoms occur.
  - 1. Spinal.
    - Locomotor ataxy.
    - Friedreich's hereditary ataxy.
    - Combined scleroses of the cord.
    - Syringomyelia.
  - 2. Peripheral nerves.
    - Pseudo-tabes.
      - *a*. Toxic.
        - Alcohol.
        - Arsenic.
        - Lead.
        - Diabetes.
      - *b*. Infectious: Beriberi.
    - "Neuro-Tabes."
  - 3. Neuroses.
    - Neurasthenic Pseudo-Tabes.
    - Hysterical Tabes.
- *B*. Diseases in which Tabetic (ataxic) symptoms are simulated.
  - Abasia.
  - Paramyoclonus Multiplex.
  - Thomsen's Disease.
  - Ataxy of Cerebellar type in
    - Cerebellar Tumours.
    - Meniérè's Disease.
    - Disseminated Sclerosis.

The author considers at length the diagnosis of these affections and gives a useful summary of the leading symptoms of each. With regard to the cases described by some authors under the title of pseudo-tabes, Dr. Blocq thinks that this name may now be abandoned, the cases really falling into two groups, the large majority being instances of paraplegia from peripheral neuritis, due to alcohol, lead, &c., the *paraplégies toxiques à type de flexion* of Professor Charcot, the others, at present comprising only a small number of cases, forming a provisional group, "*Ataxie périphèrique*" *of Déjèrine*, the exact nature of which must at present be regarded as doubtful.

J. MICHELL CLARKE.

Dana on the Cause of Chronic Degenerative Disease of the Spinal Cord.

In a paper read before the Congress of American Physicians and Surgeons (October, 1891), primary degenerations are classified as follows:—

1. Locomotor ataxia; 2. Lateral sclerosis; 3. Compound scleroses; 4. Multiple sclerosis; 5. Progressive muscular atrophy.

The recognized incurability of these affections renders the discovery of their cause a matter of great importance. The theory which Dr. Dana accepts as the most probable, is that which he terms "The Toxine Theory." Sclerosis is regarded not as a result of inflammation, a process which is now generally recognized as concerning only connective tissues and blood-vessels. The microscope indicates the chief change to be gradual decay and death of the nerve-fibre and cell. In some scleroses, as that of locomotor ataxia, this process is accompanied by the development of leucomaines or toxalbumins, which irritating substances may perhaps be charged with setting up those changes which are noticed in the connective tissue elements in this disease. Analogy certainly seems to favour the idea that degenerative processes in nerve-tissue are due to the presence of an organic poison of some sort which may be the result, either of a germ poison in the body, or of some change in the cells produced by a germ present at some previous time. The frequency with which degenerative changes follow typhoid fever, measles, scarlet fever, smallpox, and syphilis, is a circumstance which accords well with this theory.

Hock on Hæmatomyelia. (*The John Hopkins Hospital Reports*, August, 1891). Charcot and Hayem have doubted the existence of a primary hæmorrhage into the spinal cord unless produced by trauma, while Leyden, Eichorst, Gowers and Erb have admitted its existence, since it is often impossible, even with a microscopical examination, to determine whether we have to deal with a primary softening and a secondary hæmorrhage or with a primary hæmorrhage and a secondary softening, analogous to the condition in the brain. In the following cases trauma cannot be entirely excluded, still they cannot be classed under the head of the usual traumatic cases in which injury and concussion to the spine are immediately followed by a hæmorrhage, since in one case six days, in the other three weeks, intervened between the accident and the paralysis.

Case 1.—Patient was struck between the shoulders by a board falling from the height of eight to ten feet without producing any effects save some soreness next day, which soon passed off and left him quite well. Three weeks later he was suddenly seized with acute pain between the shoulders radiating down the spine and into the arms, and within *eight to ten* *minutes* by complete sensory and motor paralysis from the arms down (with the exception of the higher-situated muscles of the arm). *Retentio urinæ et alvi.* Improvement in the left leg the following day. Comparatively rapid improvement also in the right leg and left arm, so that after three weeks there remained only paralysis of some of the muscles of the right arm, especially the forearm. On examination a month after, no sensory changes but paralysis with atrophy, showing the characteristic electrical changes of certain muscles of his right arm. The muscles concerned are the following, classing them under two heads: (1) showing complete loss of motion and reaction of degeneration; extensor ulnaris, extensor communis, all the flexors, small muscles of the hand; (2) showing decrease in electrical irritability and impairment of motion; lowest part of pectoralis major and triceps. Hence, following Thorburn's scheme, the lesion is on a level with the seventh and eighth cervical and first dorsal roots, and partly upward in the sixth cervical.

Case 2.—The patient fell from a train, sustained several injuries, but no perceptible one to the spine. No trace of paralysis for six days; at that time some pain in right extremities, and after some hours' sleep complete paralysis in the right leg and arm. On examination three months after, weakness of triceps, pectoralis, flexors and extensors, and small muscles of the right hand and arm, with decrease in electrical excitability in some of these muscles; also some visible atrophy; stiffness in the right leg; no atrophy or electrical changes in muscles of leg; loss of the sense of temperature and pain on the whole non-paralysed side. There is here then a case of Brown-Sequard's paralysis resembling the picture of a syringomyelia.

The author discusses, in the differential diagnosis:—1, Hæmaorachis or hæmorrhage into the spinal canal outside the cord; 2, Acute myelitis; 3, Acute ischæmic myelomalacia, and concludes that hæmatomyelia is the most tenable from the acute onset, the quick regression of some of the symptoms, and the electrical changes. The likelihood of an antecedent sub-acute myelitis before the hæmorrhage is also discussed, but not admitted. The delay in the effect of the concussion is certainly a most interesting feature, and while the presence or absence of myelitis may be doubtful, the sudden onset of the symptoms clearly points to hæmorrhage.

Ernest Wills.

BRAIN.

PART, IV. 1893.

Original Articles.

ON A CASE OF DESCENDING DEGENERATION OF THE LEMNISCUS, CONSEQUENT ON A LESION OF THE CEREBRUM.

BY ALEXANDER BRUCE, M.A., M.D., F.R.C.P.E.

Assistant Physician to the Royal Edinburgh Infirmary.

In spite of the fact that within the last two decades considerable attention has been devoted to the course and connections of the complicated system, or systems, of fibres which are included together under the term *fillet*, or *lemniscus*, and that much has been settled by the study of its development as well as of its degeneration consequent on pathological and experimental lesions, many important points still remain obscure. The records of pathological degenerations are not only very few in number, but, with one or two exceptions, so wanting in details as to the minuter relations, that I am induced to describe the appearances in the medulla, found in a case which I have recently examined, and which seem to throw light on some of the inferior connections of the lemniscus.

The brain from which the specimen was obtained was sent me by my friend, Dr. Ruxton, of Wadsley Asylum, with the note that it had been obtained from an aged female lunatic, who had been for several years completely hemiplegic. The cause of the hemiplegia was an old hæmorrhage which had almost completely destroyed the basal ganglia and the internal capsule on the right side, and had caused considerable atrophy of the centrum ovale and of the anterior two-thirds of the corpus callosum. The extent of the lesion may be seen in the drawing, fig. 30, plate xi., illustrating my paper on the absence of the corpus callosum in the human brain (*Brain*, 1889, p. 171).

The cerebrum was at that time examined principally with reference to certain points bearing on the results of the congenital absence of the corpus callosum; while the rest of the brain, including the crura cerebri, pons varolii, and medulla, was divided into transverse slices of less than one-quarter of an inch in thickness, and put aside for future investigation. Of these parts, only the medulla oblongata is now available; the other pieces having unfortunately been mislaid. With regard to them, however, it is certain that they contained no gross lesion such as could have given rise to the degeneration to be described. The medulla, which had been carefully hardened in Müller's fluid before reaching me, was embedded in celloidin and cut into thin transverse sections. These were stained by hæmatoxylin according to the Weigert-Pal method, and also by one-quarter per cent. watery solution of aniline blue-black, as recommended by Bevan Lewis.

In the sections which were stained by hæmatoxylin, the right anterior pyramid appeared completely degenerated. Its colour was pale yellow (with the exception of certain blue fibres, to be afterwards mentioned, which traversed it), and its size, as can be seen from the drawings, was greatly reduced. Staining by aniline blue-black showed that there was a slight increase of the connective tissue. In sections made slightly above the plane of the decussation of the anterior pyramid it was easily seen that this was not the only lesion. To elucidate the other degenerations four sections have been reproduced from drawings made with great care by means of Edinger's drawing apparatus in the first instance, into which the details were subsequently filled from the microscope.

Fig. 1 is drawn from a section at the lower extremity of the medulla, immediately above the plane of the decussation of the anterior pyramid, and therefore through almost the lowest fibres which enter into the fillet.

On the right side the compact bundle of internal arcuate fibres which arises from the nuclei of both divisions of the posterior columns of the cord is seen to pass forwards, in the normal manner, as a compact, somewhat arched bundle of fibres. This, after passing outside the ascending root of the glosso-pharyngeal nerve (seen as a small mass of transversely divided fibres), turns inward and crosses the mesial plane as the decussation of the fillet (D.F.) to insert itself between the antero-lateral ground bundles (A.L.G.B.), and the anterior pyramid. (The distance to which these fillet fibres pass outwards between the two last-mentioned systems should be noted.)

On the left side these arcuate fibres are very few in number, and, at first, do not attract the eye. Those that are here present seem to be of finer calibre than those on the other side. This may, perhaps, be owing to the fact that they are few in number and separate from each other. They almost all enter the nucleus cuneatus (N.C.); of hardly any can it be said definitely that they join the nucleus gracilis (N.G.). After crossing the mesial plane at D.F. they appear between antero-lateral basis bundles and the degenerated anterior pyramid (Pyr.), with the exception of a few fibres which pass as anterior external arcuate fibres (F.A.E.A.) round the inner and ventral aspect of the pyramid (the course of which will be referred to below). Both the nucleus gracilis and the nucleus cuneatus on the left side are very considerably less than the corresponding nuclei on the right side. At this level I think that the nucleus gracilis has atrophied to a rather greater degree than has the cuneate nucleus.

The two divisions of the posterior columns seem to present a normal appearance on both sides, as also do all the other structures, with the exception, already mentioned, of the pyramids (Pyr.). There was no detectable difference in the size of the ascending roots of the 5th nerve, the substantiæ gelatinosæ (S.G.), the direct cerebellar tracts (D.C.T.), the antero-lateral ground bundles (A.L.G.B.), or the anterior external arcuate fibres (F.A.E.A.). The asymmetry of the two sides of the section is due to the shrinkage of the degenerated right anterior pyramid, and of the lower end of the fillet, which has disappeared from the spot immediately behind it.

Fig. 2 is drawn from a section made through the lower extremity of the inferior olive. On the right side, the nucleus gracilis and nucleus cuneatus are of normal size. The median internal arcuate fibres (F.A.I.M.), which emerge from these, pass forwards and inwards in curves with their convexity outwards. They may be almost divided into two groups, a posterior and an anterior. The posterior group forms a bundle of fibres nearly as compact as in fig. 1. Some of these pass close round the inner side of the ascending root of the glosso-pharyngeal nerve (and indeed seem to arise from it). This group of fibres passes through the more posterior fibres of the posterior longitudinal fasciculus, crosses the raphe at a somewhat acute angle, and passes forwards and slightly outwards. The second or anterior group of these internal arcuate fibres passes forward through the formatio reticularis in more open curves of somewhat widely separated fibres, which cross the raphe and enter the fillet (or inter-olivary stratum) of the opposite side.

On the left side the two nuclei of the posterior columns are atrophied, apparently to nearly equal degree (though on the whole, I think that the nucleus gracilis is the more affected). The fine felt work of fibres within these nuclei has to a large extent disappeared, so that their colour in hæmatoxylin sections is decidedly paler than those on the right side. The median internal arcuate fibres have almost entirely disappeared from this side. Those which pass to the inner side of the ascending root of the glosso-pharyngeal nerve have all but entirely gone. Of the others, a small number of separate (and fine) fibres arch along the dorsal part of the formatio reticularis, and cross the posterior longitudinal fasciculus to enter the inter-olivary stratum of the opposite side. The comparison of the formatio reticularis in the intermediate field of Flechsig on the two sides is most striking. On the right the arcuate fibres form a distinct tract, which catches the eye at once, on the left there remain but a few fibres which require a careful search to be seen. The contrast between the two sides of Flechsig's inner field, viz., that between the raphe and the root of the hypoglossal nerve (not figured) is no less striking, though perhaps not quite so much so as in figs. 3 and 4. The strip of transversely divided fibres which extends along the right side of the raphe from the hypoglossal nucleus to the degenerated anterior pyramid is greatly reduced in size. This change is most marked anteriorly, where the fibres become attenuated to a narrow point (Fill.) which projects into a pale V-shaped area, the outer limit of the V being the internal accessory olive; and the inner, the area from which the degenerated fibres of the fillet have disappeared. The most dorsally situated fibres in the area form a band considerably less broad than the corresponding ones on the other side. The anterior external arcuate fibres which are seen to arise from the fillet, pass, partly as a small strand between the inferior olive and the degenerated pyramid, partly round the inner and anterior aspects of the pyramid, at the outer side of which it joins the first strand. A third series passes as scattered fibres into the pyramid. I have been unable to detect any diminution in the number of any of these three strands of external arcuate fibres on careful comparison of the two sides. Beyond the asymmetry of the medulla, which results from the above-mentioned degenerations, no other abnormality could be seen in the section.

Fig. 3 represents a plane of section very slightly higher than that of fig. 2, but still through the lower end of the olive. The asymmetry of the two sides of the section is as marked as in fig. 2, and its cause is the same. The all but complete disappearance of the internal arcuate fibres on the left side is as marked as in fig. 2. Those fibres which remain are fine and separate from each other. They lie almost entirely in the posterior part of the formatio reticularis, and terminate mainly in the internal cuneate nucleus, but also partly in the nucleus gracilis. The cuneate nucleus appears in this section to show a relatively rather higher degree of atrophy than does the nucleus gracilis. Both are much paler than normal, owing to the disappearance from their interior of the terminal twigs of the arcuate fibres. In Flechsig's inner field the contrast between the two sides is greater than in fig. 2. The fibres in this region which lie between the hypoglossal nucleus and the anterior pyramid are usually divided into two pretty nearly equal parts, a line drawn between the two dorsal ends of the internal accessory olive forming the supposed boundary line. The portion of fibres behind this imaginary line is usually regarded as the posterior longitudinal fasciculus, that anterior to it as the fillet (or inter-olivary stratum). Both these strands are reduced in breadth, but to different degrees. The posterior half is about one-half the width, the anterior is about one-eighth or less, being fined away to a narrow point anteriorly, so that the internal accessory olive comes almost to touch the raphe. A fine pale line is seen along the outer side of both halves of the strand, showing where the degenerated fibres have fallen out. The hypoglossal nerve on the right side has approached the raphe considerably, being only about half as far from it as the left nerve.

The external anterior arcuate fibres which lie in front of and behind the right anterior pyramid, are in no way fewer than those of the opposite side. The section was otherwise perfectly normal.

Fig. 4 was drawn from a section made slightly above the middle of the olive, and just above the upper end of the nucleus gracilis. On the left side those median internal arcuate fibres which pass from the cuneate nucleus behind the inferior olive, have almost entirely disappeared. A few can still be seen to enter the internal cuneate nucleus (N.C.I.), but those which, on the right side, pass in relation to, and as if arising from, the ascending root of the glosso-pharyngeal nerve (IX. Asc.) have entirely disappeared. On the right side all the median internal arcuate fibres are normal. The lateral internal arcuate fibres which pass from the left restiform body, partly through, partly behind, and partly in front of the olive, to end in the opposite olive, are equally present on the left as on the right side. The great contraction of the fibres in the whole extent of the inner field of Flechsig is very distinct, being marked off from the rest of the formatio reticularis by a narrow pale band, which widens out anteriorly, so that in the drawing it is difficult to distinguish it from the internal accessory olive. The posterior part, which corresponds to the posterior longitudinal fasciculus (P.L.F.), is, on the right side, only one-half the breadth of that on the left; while the inter-olivary stratum (Fill), is reduced to one-eighth of the breadth of the other. Those fillet fibres which are found between the internal accessory and the main olive, have also disappeared on the right side. The anterior external arcuate fibres (F.A.E.A.)

are quite normal on both sides. The two restiform bodies (C.R.) are quite equal, and the posterior external arcuate fibres (F.A.E.P.) are in no way diminished on the left side. There is no other abnormality in the section.

This case shows that, in consequence of a lesion destroying the basal ganglia and the internal capsule in the cerebrum, the fillet may degenerate in a descending direction, through the inner field of Flechsig in the same side of the medulla, through the median internal arcuate fibres of the opposite side, as far as the nuclei of the two divisions of the posterior columns of the cord.

The degeneration in the inner field is not co-extensive with what is usually regarded as the inter-olivary stratum. This name is given to those longitudinal fibres which are contained between the raphe and the inferior olive, and between the posterior surface of the medulla and an imaginary line joining the hinder limits of the two internal accessory olives.

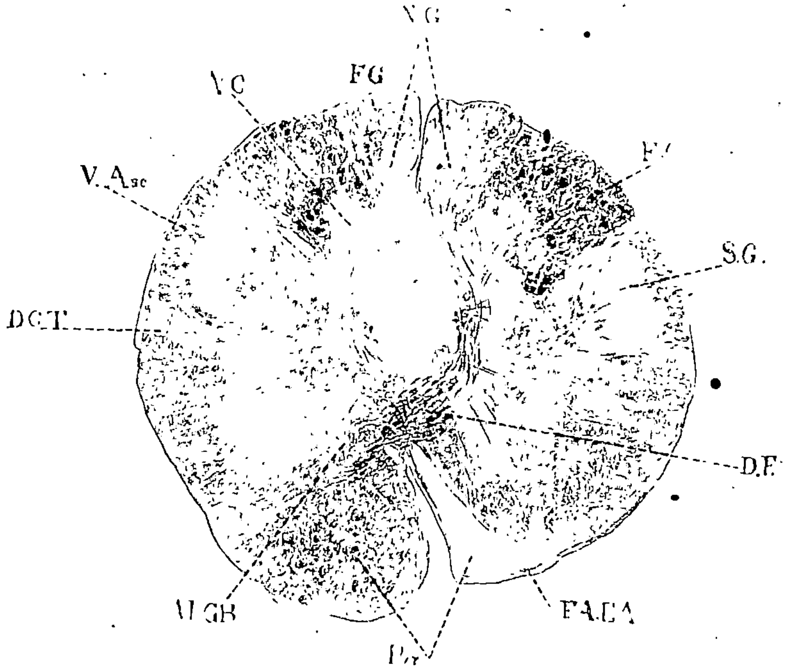
The degeneration affects about seven-eighths of the fibres in this area, those fibres which remain intact forming a very narrow zone along the side of the raphe. But the degeneration also extends in a backward direction as a very narrow zone along the outer side of the posterior longitudinal fasciculus. From the examination of several other cases of degeneration in the medulla, I can find no evidence to show that these fibres do not belong to the fillet, and I am inclined to think, therefore, that the limits of the fillet are more extensive than developmental evidence would appear to show.

Those fibres which remain in the anterior part of the inner field are either part of the system of the posterior longitudinal fasciculus, or are fibres of the fillet which have escaped degeneration, either because they degenerate only in an upward direction, or, if they degenerate downwards, because they arise in the pons and corpora quadrigemina, from levels too far down to be affected by a lesion in the cerebrum. We shall see reason to believe that the latter is the case. It is possible that this may be the explanation, but it would be of great importance to compare this case with one where a descending degeneration had followed a pontal lesion, and where presumably both the cerebral and pontine fillets had been cut across. In Spitzka's case (*loc. cit.*) the whole stratum would appear to have degenerated, but his figures are somewhat too small to enable one to form a definite opinion from them.

Certain of the internal arcuate fibres of the opposite side have disappeared. The lateral group which connects one olive with the opposite restiform body, are quite intact; but the great majority of the median arcuate fibres which are found in the lower part of the medulla have disappeared. A comparatively small number remains. These are seen as fine fibres, extending from the raphe along the most posterior part of the formatio reticularis, towards both nuclei of the posterior columns.

At the lower part of the medulla the number increases, till a compact bundle is formed, which can be traced pretty definitely for the most part from the undegenerated remnant of the opposite fillet into the nucleus cuneatus. It would appear from this, therefore, that the nucleus cuneatus is more directly connected, than is the nucleus gracilis, with those fibres of the fillet which end below the cerebrum. This is in harmony with the results obtained from the embryo (see Illustrations of Mid and Hind Brain, plate xiii., fig. 3, where it is seen that the earliest fibres of the fillet arise from the cuneate nucleus, and cannot be traced higher than the pons; see also reference to Bechterew's paper on the Fillet at page 37, which agrees with this view). It is certain, however, that all the persistent arcuate fibres do not arise from the fillet, but that a large number of them are directly continuous with the opposite anterior external arcuate fibres, which pass in undiminished numbers to the opposite restiform body, partly in front of, partly through, and partly behind the anterior pyramid. Hösel says that in his case they have entirely disappeared from within the pyramid. I have carefully compared my sections with others from a simple degeneration of the pyramid, and can find no difference in the numbers of undegenerated fibres within the pyramids in the two cases. It is possible that Hösel has over-estimated the number of these fibres in the sound side, as it is likely that the wavy appearance, noted by him as due to the passage of arcuate fibres through the pyramid, is largely the result of a re-arrangement of the pyramidal fibres, preparatory to their decussation. It should be noted also that

Fg I


N.G
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Fg II

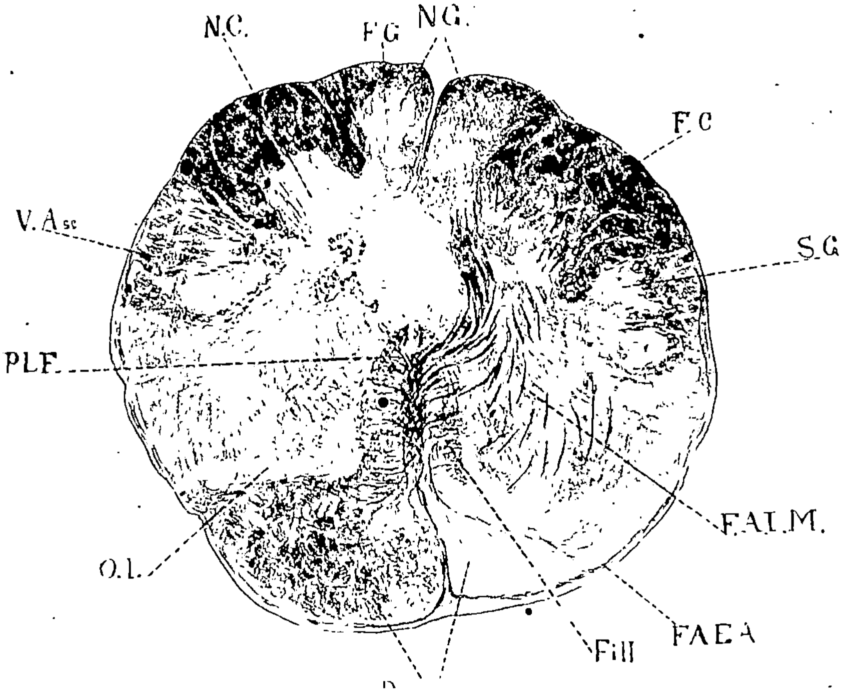
N.C.
F.G
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Fig III

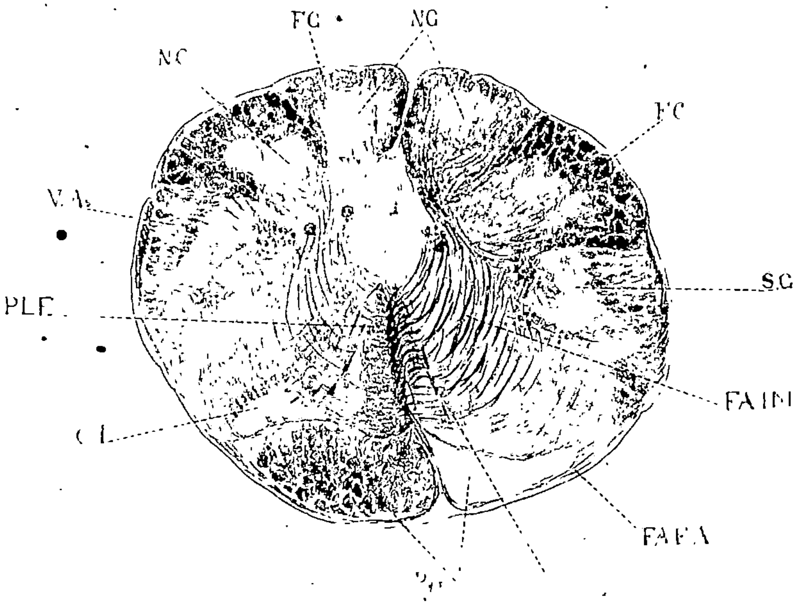
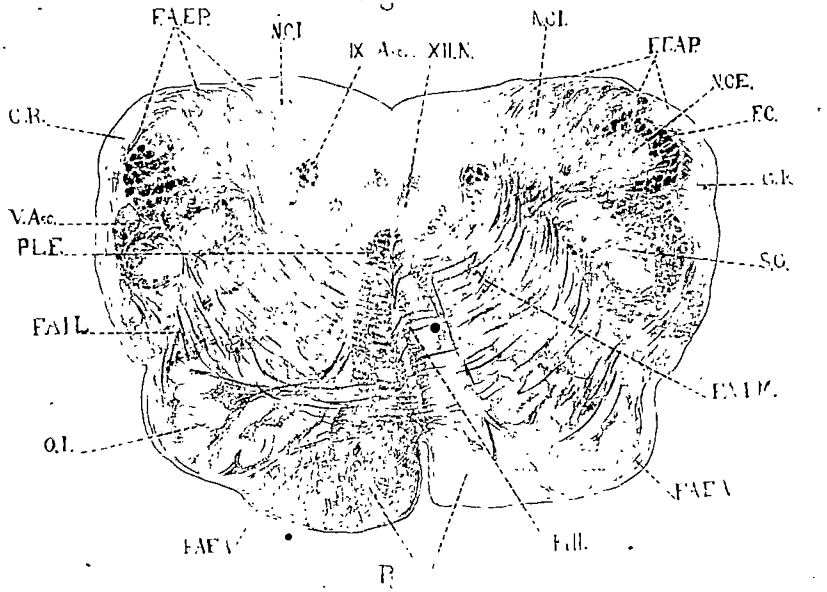


Fig IV



those internal arcuate fibres which pass from the inner side of the ascending root of the 9th nerve, have all but disappeared, while the nerve itself is quite normal. Through the kindness of Dr. Abernethy I have had the opportunity of comparing with this case one of degeneration of the ascending root of the 9th nerve, in which these arcuate fibres are intact. It is, therefore, certain that the ascending root of the glosso-pharyngeal nerve is not directly connected with the fillet.

Both nuclei of the posterior columns showed a considerable degree of atrophy on the left side. This was evidenced, in the hæmatoxylin section, by a decrease in the size of the nuclei, and a diminution of the network of fibres belonging to the internal arcuate system; and, in sections stained with aniline blue-black, by disappearance of the nerve cells and also by an increase of the connective tissue. At the lower extremity of the medulla, the nucleus gracilis seems to have atrophied to a greater degree than the nucleus cuneatus; but at levels opposite the upper extremity of the nucleus gracilis the internal cuneate nucleus seemed to have degenerated to an equal, if not to a greater, extent. These facts would appear to warrant the following conclusions as to the connections of the fillet: that the nucleus gracilis is the terminal point for the great majority of the fibres which pass upwards to the cerebrum, while the nucleus cuneatus, at its lower part, receives mainly fillet fibres from the pons, which have escaped degeneration, but, in its upper part, getting also fibres from the cortical fillet. Observers of the degeneration of the nuclei have obtained somewhat divergent results; Monakow finding in an experimental case that the nucleus gracilis was the more atrophied, while Déjérine, Flechsig, and Hösel found the greater change in the cuneate nucleus. Spitzka, in his case, where the lesion was in the pons, and where, presumably, all the fibres of the fillet were degenerated, found the nucleus gracilis the more affected. The external cuneate nuclei are unaffected, and are therefore, presumably, not end stations of the fillet at all.

It is remarkable that none of the other tracts connected with these two nuclei show any evidence of diminution in size. Both divisions of the posterior columns of the two sides are equal, so, too, are both sets of posterior and anterior external arcuate fibres.

It follows, therefore, either that there are in these nuclei still undegenerated cells, which are the trophic centres for these arcuate fibres, or else, that their trophic centre is situated in the cerebellum. My case does not enable one to draw any conclusion as to this, which can only be proved by a comparison of destructive lesions in the nuclei or in the middle lobe of the cerebellum, the two terminal points of these strands. It is known that the posterior columns do not degenerate downwards.

Unlike Hösel's case, no difference could be traced between the two 5th nerves.

The most important cases of descending degeneration from a cerebral lesion are those of Witkowski (*Arch. f. Psychiatrie*, Bd. xiv., p. 140); Déjérine (*Arch. de Physiol.*, 1890); Hösel (*Arch. f. Psychatrie*, Bd. xxiv., p. 452); Monakow (*Neurol. Centralblatt*, 1885, p. 69). Spitzka, in the *Medical Record*, 1884, p. 393, describes carefully a descending degeneration from a lesion in the pons. Most of the other cases, which are fully referred to in Déjérine's and Hösel's papers, are too briefly described to be of much value.

The four figures are photogravures (by Messrs. Thevoz, of Geneva) of the original pencil drawings. Fig. IV. is reduced in scale ($\frac{8}{11}$).

REFERENCES IN FIGURES.

A L G B, antero-lateral ground bundles.
C R, restiform bodies.
D C T, direct cerebellar tracts.
D F, decussation of fillet.
F A E A, antero-external arcuate fibres.
F A E P, postero-external arcuate fibres.
F A I M, medio-internal arcuate fibres.
Fill, fillet (not lettered in fig. 3).
N C, nucleus cuneatus.
N C I, internal cuneate nucleus.
N G, nucleus gracilis.
O I, inferior olive.
P L F, postero-longitudinal fasciculus.
Pyr, anterior pyramids.
S G, substantia gelatinosa.
V.Asc., IX.Asc., XII.N., v., ix. and xii. nerves.

A THEORY OF CORTICAL VISUAL REPRESENTATION.

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BEFORE attempting any theory of the representation of vision in the cerebral cortex that might explain all the opposing facts, it would seem necessary to enumerate a few of the most important, the best authenticated, and it would also appear *pari passu* most contrary of the results of and deductions from experiments on animals in this relation. For while, like all other kinds of cerebral localisation, that of the cortical centres for vision owes its discovery to experimental investigation, in this particular branch of this method of research so many different inferences have been drawn from results which in themselves have been of such varied and often contrary nature, that even after a careful study of the literature of the subject it is difficult to decide which, if even any, are now regarded as conclusive and indisputable.

And first as to the mere extent of the visual area. It would seem that in the opinion of many this comprises both the angular gyrus and the occipital lobe, for Ferrier states "in order to cause complete and permanent loss of vision in both eyes it is necessary completely to extirpate both angular gyri and both occipital lobes" ("Functions of Brain," p. 272), and again that "complete destruction of this area (the occipito-angular) in the one hemisphere causes permanent hemiopia to the opposite side . . . . while bilateral destruction causes complete and enduring blindness of both eyes" ("Cerebral Localisation," p. 40), where he goes on to state (pp. 46-48) that Tamburini and

Luciani too include the angular gyrus in the visual area; and Schäfer and Horsley also appear to believe that the angular gyrus is to some extent concerned in vision as well as the occipital lobe.

To many an unbiassed view of Munk's own account, from his description of the phenomena noticed after lesion of this part, would lead to the same inference, though he himself professes to confine the visual area to the occipital lobe alone. Certainly, the movements of eyeballs and pupils, and sometimes of the head, which occur on stimulation of this region, and which, considering the length of the latent period, can only be interpreted as reactions to a sensory stimulus, supports the view of the area's function; so that I think we may fairly say that there are good grounds, without discussing the clinical side of the question, for believing that the cortical visual area probably includes the angular gyrus as well as the occipital lobe.

Considering each of these two regions separately; as to the occipital lobe, it is well known that many observers—Schäfer, Munk, Horsley and Schäfer, Sanger Brown and Schäfer, Erlman, Thompson and Sanger Brown, with others—describe a condition of homonymous hemiopia from paralysis of correlated halves of the same-named sides of the retinæ, after cortical lesion of this alone, and some profess to show blindness following a bilateral lesion. On this point there is not agreement, however, for Horsley and Schäfer also show that after removal of both occipital lobes some vision remains, and though a large amount of clinical evidence has been brought to prove the pre-eminent visual function of this lobe, still the fact remains that many cases, also of lesions of the occipital lobe, have been reported without any observable symptoms (as instanced by Ferrier's "Localisation of Cerebral Disease,") and though, if unilateral, this might be accounted for from the hemiopia being unnoticed or overlooked—as, indeed, it often is in actual clinical practice — still, as these cases include some of bilateral lesions when the patient should theoretically have become blind, it is obvious that some other explanation is necessary. Schäfer himself (BRAIN, vol. x., p. 372) states that a lesion to produce permanent hemianopia from injury to the occipital lobe probably must include some at least of the cortex of the angular gyrus, and this would agree with Ferrier's hypothesis that by so doing a portion at any rate of the angular radiations are involved, and I hope to be able to show that for the blindness *per se* seen after bilateral lesions this is probably the explanation.

Ferrier has laid it down that "even after most extensive destruction of the occipital lobes no portions of the retinæ appear to be absolutely blind," by which I conceive that the animals still retained some vision, and he also states ("Functions of Brain," p. 273) that the occipital lobes *per se* "can be injured or cut off bodily almost up to the parieto-occipital fissure on one or both sides simultaneously without the slightest appreciable impairment of vision."

Lannegrace (*Archives de Médecine Expérimentale*, 1889) has also published results of experiments in which he too failed to obtain the impairment of vision spoken of by other observers, so that it is evident, without referring to the clinical side of the question, which will be returned to later, that some observers still doubt the occipital lobe being at any rate the sole and only cortical area for vision, but that there is much evidence in favour of it taking a large share seems certain.

As regards the angular gyrus alone, Ferrier has from the first laid it down that unilateral lesions of the angular gyrus cause temporary blindness in the opposite eye, giving at the same time reasons for believing that the eye of the same side was simultaneously, but to a much less extent, affected. Also that bilateral lesions caused blindness of both eyes, though this too was only of a temporary character.

Lannegrace also found crossed temporary amblyopia to follow a lesion of this area, and Schäfer (BRAIN, 1888, p. 159), after destroying both angular gyri found that blindness for a few days followed. So that it would appear that the angular gyrus is also credited with a considerable share of cerebral visual perception, though in this case of a totally different character to the function of the occipital lobe.

As a result of all these conflicting observations several equally conflicting deductions have been generally accepted.

(1) That the visual area on each side comprises both angular gyrus and occipital lobe.

(2) That hemiopia follows lesion of the occipital lobe, (*a*) according to some, only when associated with damage to fibres from the angular gyrus; or, according to many, (*β*) after lesions of this lobe alone and of that only, Schäfer, as is well known, in his scheme also providing for a (*γ*) sectional representation of the visual field, in the occipital cortex.

(3) That in the angular gyrus of each side there is a "higher" representation of the whole of the opposite visual field, (*a*) and to a smaller extent of that of the same side (Gowers). (*β*) That this area (angular gyrus) probably, as Ferrier has shown, corresponds to that for clearest vision, *i.e.*, central vision as subserved by the macula lutea.

It will, therefore, be evident that these facts and theories necessitate that whatever scheme would really explain and reconcile all these differences must account for the following:—

(1) The angular gyrus and the occipital lobe being both concerned.

(2) Amblyopia of a temporary character following experimental unilateral lesions of the angular gyrus, and bilateral lesions of the same producing blindness also of a temporary character.

(3) Unilateral occipital lobe lesions being followed at some hands by homonymous hemiopia and bilateral lesions by blindness; while at other hands, unilateral and bilateral were both equally productive of negative results.

(4) Smaller occipital lesions showing instead of complete hemiopia some sector defects.

(5) The evidence in favour of the angular gyrus subserving central vision, *i.e.*, the area of clearest vision and the yellow spot.

Now there is one theory of cortical visual representation which it seems to the writer would account for all the perplexing contrary experimental results, would reconcile these several conditions, and would also agree with several clinical facts hitherto difficult to account for.

I would venture to submit that vision in the general area of the retina is distinct from that of the macula lutea, *i.e.*, that though both may frequently, and, indeed, usually may, act together, vision is possible by the retina alone without the macula lutea; or, on the other hand, by the macula lutea alone without the surrounding retina. For it is laid down and generally accepted that for clear sight, putting aside the question of the size of the visual angle, the state of the cornea, lens, vitreous, and amount of illumination and pupil area, the conditions are that the image, as well as being focussed on the retina, must be formed at the centre of the yellow spot, and it will not, I think, be denied that it is the yellow spot that subserves any and every occasion for clearest and most distinct vision. But given a perfectly normal macula lutea in ordinary use, it is almost equally certain that the remaining area of the retina, even allowing that the extreme outer periphery may not do so (Landolt), also subserves vision; as to this fact it seems almost needless to bring evidence, but there is certainly a marked loss in such diseases as syphilitic, hæmorrhagic and pigmented retinitis, the defect of sight being often in direct ratio to the amount of retina affected—until only the central area subserved by the macula lutea may remain, and it is here worthy of note that vision may be exceedingly good in a bright light when only this portion remains to see with.

On the other hand, axial neuritis and tobacco atrophy may both cause a distinct loss of central vision with a well-marked central scotoma, but still a very fair amount of vision is retained by the unaffected surrounding retina. Indeed, in this particular I would remark that a central loss of sight may be quite unperceived for some time. We are none of us, I think, aware of the blind spot normally present in our retinæ, and the central scotoma of tobacco amblyopia is occasionally overlooked by its possessor. A good deal, perhaps, depends on the intelligence and power of observation of the patient, which, however, does not affect the fact, so that we may allow that vision is possible with loss of the centre of the field if there is retention of the periphery, and that it is equally possible with the periphery lost or damaged and the centre remaining. It is probable that most persons, unless for, if I may so term it, loose general vision, use the macula lutea, chiefly if not exclusively for vision requiring any attention or application, and it would presumably follow that its loss would be very evident. Now, I would therefore suggest that the true explanation of the discrepancies of experimental, and indeed clinical evidence lies in the fact that there is a separate and distinct representation of the macula lutea and the correlated retinal halves in the cortex, namely, that the macula lutea and that only is represented in the angular gyrus, chiefly of the opposite side, but to a smaller extent of the same side also; the representation being the bilateral one enunciated by Ferrier ("Cerebral Localisation," p. 58), and now largely accepted; while each occipital lobe serves for the representation of the correlated halves of the same named side of each retina, but without the macula lutea of either, which is represented solely in the angular gyrus, as before said.

On consideration as to how this theory would account for the opposing results of experiments, &c., taking first the most striking, *i.e.*, the negative effects of occipital lobe lesions, in accordance with this scheme, a lesion strictly confined to the occipital lobe would then cause a hemiopia to the opposite side, but there being still distinct vision in both maculæ luteæ, which certainly in animals is the portion of the retina chiefly used in perception of objects, &c., by which the animal's sense of sight is generally tested in the laboratory, the fact of there being much loss or any could easily escape observation; and even in a bilateral lesion given it still strictly confined to the occipital lobe even then blindness would not result, both maculæ luteæ still remaining, while on the other hand the extreme ease with which the angular radiations might be injured in the experiment would account for the very complete hemianopia so distinctly described by many observers, and *a fortiori* for the blindness they have seen to follow bilateral injuries.

The occipital cortex, in my opinion, particularly subserves crude binocular vision; and I think this lobe will be found developed in animals in direct ratio to the amount of binocular vision they possess, while the angular gyrus is the "higher" field of representation of particular clearest and central visual perception. It is not, of course, pretended for a moment that the macula lutea cannot and does not subserve binocular visio[illegible] it undoubtedly does, but that is not its essential function, [illegible]ainder of the retina, it is maintained, being the essent[illegible]nocular mechanism.

In regard to the amblyopia see[illegible]er experimental lesions in the angular gyrus, the macula l[illegible]a and that part only being represented in this area in such a manner that each angular gyrus subserves the macula lutea of the opposite side, and to a less extent of its own, and this being in animals as in men such an important part of the visual field, the disturbance resulting from a sudden loss of it such as would result from an experimental lesion would, I think, satisfactorily account for the temporary amblyopia that has been seen to follow it, and the fact of its being only temporary would, it is obvious, be accounted for by its bilateral representation, and perhaps to a smaller extent by the gradual use of the peripheral retina, which latter fact would be the explanation of the non-permanence even of bilateral lesions.

From an anatomical point of view a scheme to represent this theory could easily be given, for, in addition to the crossing in the chiasma of the fibres from the inner half of each retina, all that would be required would be to show the fibres from each macula lutea also crossing in the chiasma with these and going with them, and the temporal fibres of each side, after communication with the optic thalamus, the internal geniculate and anterior quadrigeminal bodies, as the optic radiations, separating and passing to their several distributions in the angular gyrus and the occipital lobe shortly after. But to account for the double representation in the angular gyrus almost undoubtedly present, some fibres from each macula lutea must also be continued in the optic tract of the same side to the cortex, and though another possible arrangement in place of this, *i.e.*, that of fibres being continued along the same side, is that which has been supposed in nearly all the schemes hitherto brought forward, namely, that a re-crossing of some fibres at least takes place in the mid-visual centres (presumably the quadrigeminal or geniculate bodies, for the fibres going to the optic thalamus almost undoubtedly subserve "hemiopic" vision), still, it is generally stated that the decussating fibres over the anterior quadrigeminal bodies crossing the aqueduct, if not solely, are largely derived from the fillet, others from the "central connections of the descending root of the trigeminus" (Obersteiner); and though the posterior quadrigeminal bodies are almost continuous with one another in the middle line, still these latter have been generally believed not to be related in any way to vision *per se*, and as I am certainly not aware of any one having shown a decussation of the geniculate bodies it would seem as though this arrangement in the chiasma were the only possible one, although at the same time the bilateral representation could be more easily understood by a division of the fibres after their junction with cells (*i.e.*, that cells receiving one apparent process could give off two, one to be reckoned again for distribution to the opposite side), than it can be understood how a double set of fibres should go from identically the same area in the macula lutea.

It seems needless to remark that this is not a solitary example of a bilateral representation in the cortex—the same being seen so conspicuously in that of the trunk muscles, respiratory, laryngeal, and all these habitually used in concert, and though it might appear strange that so small a portion of the retina or the macula lutea should have a separate representation in this way, it may be noticed that apart from the functional importance—from the extreme tenuity of the fibres an extremely large number are distributed to this area. Bunge (quoted by Nettleship, p. 199) gives a case of central scotoma, in which though only onefortieth of the field was lost, yet one-fourth of the whole of the fibres were degenerated. There is probably, too, a difference histologically as well as in number between the fibres of the retina and the macula, for it has been repeatedly shown that fibres from some part of each optic nerve pass into both tracts, the probability being that these are from the fovea, and attempts have been even made to show that degeneration which has been traced into both tracts is from this area. Gowers (vol. ii., p. 142) seems, while doubting this latter particular, to take it for granted that fibres from each macular region pass to both hemispheres, but does not give any details; and Obersteiner ("Anatomy of Central Nervous Organs," p. 284), while he says "that each visual centre is connected in a partially crossed manner with both eyes," also does not explain how this takes place; certainly no one has ever even suggested that the "hemiopic" fields are ever doubly represented, *i.e.*, that any part from the same named halves are represented in both hemispheres, so that it is altogether more feasible to suppose these double fibres to be derived from the central fovea, and it is just possible that the "direct cortical root," that is to say, the deep root, of fibres branching off from the tract to the crus in front of the geniculate bodies, and which, as Obersteiner remarks, probably contains fibres from both optic nerves, is the path of these macular fibres to the cortex. If this were so, one would expect to hear of direct degeneration between the angular gyrus down this tract to the optic nerve, or *vice versâ*, there being no cells (?) midway to break the process. As to the functional representation of the angular gyrus and occipital lobe respectively, it seems probable that the representation in the angular gyrus is as of a whole, *i.e.*, is not a functional division into segments, so that any partial lesion would only cause a partial lowering of the whole functional capacity and a relative loss; whereas in the occipital lobe it seems most probable that there *is* a segmental functional division; whether according to Schäfer or Munk's scheme, to which or to either, is not clear. Arguments have been brought forward that the cases adduced to prove this clinically are always examples of implication of the optic radiations and not of disease of the cortex, and though this fact in these instances is more than probable, still, it is difficult to understand why differentiated optic radiations should exist just under the cortex if not for distribution to different parts of that cortex, it being admitted that these different radiations are in relation with segments of the retinal area. A careful consideration of defects of sight from a clinical point of view show facts which agree with this theory as to the separate representation of the centre and the periphery of the retina in the cortex of the hemispheres—facts which may also serve to explain certain points in connection with them hitherto of very debatable character.

It was first shown by Willbrand as a distinguishing mark between tract and central hemiopia, that a pencil of light thrown on the blind side of the retina failed in the former variety to cause contraction of the pupil. But Ferrier (" Functions of the Brain," p. 294) has also shown that all these cases have the fixation point cut by the dividing line. Now everyone is aware of the extraordinary difference in many cases of hemiopia, whether the dividing line passes through the fixation point, or whether this is included in the seeing half (Willbrand puts the variation as 23 in 50), that is, whether the macula lutea region is divided or not. Gowers (" Manual," vol. ii., p. 142) tries to account for this by doubting whether the fixation point is ever actually divided, but it must be obvious that according to his argument when the line is, as he shows it, so extremely close to the fixation point in division, the probability of an error is enormously increased. I mean that if a patient first denies seeing beyond the fixed area, and then, being brought down to the division of a minute point, inclines to think that he sees it—considering the difficulty at all times of keeping the line of sight motionless—it is extremely probable that his first view was the correct one.

I would, therefore, submit that the true explanation lies in the distribution of area suggested, namely, that in the tract, of course the macular fibres still flowing with those of the other part of the retina; a hemiopia due to lesion here which would necessarily include these too would not show a retention of the centre in the seeing half of the field. Whereas, after the individual centres—the optic thalamus, the geniculate, and quadrigeminal bodies, the macular fibres going to the angular gyrus diverge from the retinal ones proceeding to the occipital lobe, and therefore an affection directly in or under the cortex of the latter would cause a hemiopia, in which the fixation point was included in the half in which vision was still retained. I am aware that it would seem at first that if this explanation holds, then where the fixation point is included in the blind half that there should be a small projection of it into the seeing portion to agree with the fact that the macular fibres are not divided, but decussate as a whole. I have not had an opportunity lately of testing this point, but I do not think the line is ever perfectly straight, and the area is so small that it might easily escape observation. At the same time it must be remembered that if the lesion were sufficiently low down, near the optic tract, where presumably the macular fibres would be only just leaving the company of the others if they were not indeed still in conjunction, the hemiopia would simulate that due to disease of the tract itself. This would account for the apparent variability of their symptoms and for Gowers' dictum that the difference in the fixation point does not depend on the situation of the lesion. I would urge that it most certainly does, and that a careful examination of cortical and subcortical cases of hemiopia where the angular gyrus is not involved, will always show that the fixation point is in them included in the seeing half. Several of the figures Gowers himself gives, and which presumably are of cortical disease, show this, viz.; figures 75, 76, 77, 80. I have never seen a segmental defect illustrated (which is, *par excellence*, usually supposed to be due to a lesion of the occipital lobe, and if not cortical, subcortical) in which the central area was represented as blind, which is a fairly strong argument.

In those cases where the fixation point is divided, and yet the lesion is not in or near the tract, I think it will be found that there is generally at the same time a peripheral contraction of the remaining half fields, and that, as might be expected, there is usually some implication of the fibres radiating from the angular gyrus.

The clinical cases of occipital lesions in which there were no visual symptoms would probably be all explained by the assumption before given for corresponding experimental results, that the angular gyrus being unaffected some sight did actually remain. But still, there is very strong clinical proof of the association of occipital lobe disease (either alone or plus that of the angular gyrus) and hemiopia, cases even of amaurosis after bilateral lesions having been reported. As to there being any evidence of actual cortical disease producing such is another matter. I had occasion to examine carefully the original records of all the recorded cases of occipital and parietal lobe lesion up to June, 1890, and failed to find a single case of cortical lesion strictly limited to the occipital cortex causing hemiopia. Nearly all the many described as coming under this category, even when they were not due to or accompanied by a tumour or compressing lesion in the medullary substance, in which case, for localisation purposes, they are of course absolutely valueless, even when not so were each accompanied by so many other lesions in the temporal hippocampal or parietal cortex as to render it absolutely impossible to say that the symptoms were due to affection of the occipital lobe alone. It has also been strongly argued by Seguin and Nothnagel that the cuneus is the ultimate seat of hemiopic visual perception, and Obersteiner in his recent work also places this as the culminating point in the visual scheme. It may be so, in which case it is difficult to see the utility of so large a remaining portion of the occipital lobe, but the cases brought in support of this theory are open to the same objections before alluded to, and I am not aware that any one has ever proved the optic radiations to end at this particular point, although it is most probable that the implications of these radiations is the explanation of these cases, and also of that theory (Exner) that would substitute the apex for the cuneus. In monkeys too it may be remarked the cuneus may be excised without appreciable result as to vision.

As regards the evidence on the clinical side, *i.e.*, the angular gyrus, there are now a fair number of cases, some with *post-mortems* in which loss of sight, not of retinal halves, but an amblyopic defect in one eye has been shown to depend on a lesion of this area on the opposite side to which the principal long sight exists, there being always a smaller loss in the eye of the same side also. (Ferrier, BRAIN, vol. iii., p. 456, and "Gowers," vol. ii., p. 19, and Sharkey, *Med. Chir. Trans.*, 1884, p. 265.) So many cases are now on record that Gowers' theory ("Manual," p. 19) of there being a "higher visual centre" in front of that for the half fields has been very generally accepted, and Ferrier's dictum as to this point being the centre for distinct vision ("Cerebral Localisation," p. 58), *i.e.*, from the macula lutea, has been shown to be most highly probable. I would therefore venture to admit that the macula lutea is represented in this area exclusively, chiefly of the opposite eye, and to a smaller extent for the same side as that of the lesion causing crossed amblyopia. But in this particular, there is one fact somewhat difficult to account for, namely that in all these cases reported the clinical symptom has been a peripheral restriction of the field, and not a loss of central vision as one would have expected. The cases have mostly been so carefully reported that a mistake is scarcely possible, and on the other hand it is almost without doubt that the loss is one of central vision; it is difficult to reconcile the discrepancy, but the facts are so distinct that one must be contented with simply finding the explanation, and I can only suggest that as the representation is bilateral, and that therefore the opposite hemisphere also subserves this very area, *i.e.*, the fovea, it is that which is retained though much lessened.

A series of cases have been reported shewing that lesions of the angular gyrus of the left side cause word blindness, and the relation is now fairly established, as also is the fact that the affection is not of necessity accompanied by any affection of vision in its ordinary sense; if there is any one occupation *par excellence* requiring the use of clearest vision and therefore of the yellow spot, I should imagine it to be that of reading, so that this symptom following a lesion of this area is scarcely to be wondered at, and its cortical limitation to the left side is only a further amplification of the general rule in that respect as regards all the other varieties of aphasia. As word blindness is not necessarily accompanied by visual loss, though it should accompany a right-sided amblyopia, theoretically, its explanation is probably that of a partial lesion or the partial and remaining effect of a lesion otherwise compensated for showing itself in a loss of those faculties latest acquired, visual ideation suffering more readily and more lastingly than simple visual perception.

The same may be said of the cases of mind blindness described as occurring clinically, for example, those of Chauffard (*Rev. Med.*, 1881, p. 440), and MacEwen (*British Medical Journal*, 1888, p. 306), and possibly of the similar condition described in the lower animals by Munk and Goltz. If the cell modifications which presumably occur as a result of a stimulus, *i.e.*, a perception, being, as it is reasonable to suppose they are, permanently impressed there or bearing an "impression" and so constituting in each a residual path of lessened resistance and thus in each sensory centre, the organic basis of the memory of its own sensory impressions one can understand, even without resorting to the cumbrous theory of a portion of the cortex being reserved for the storing up of visual images, the fact of the possibility of a lesion here causing inability to recognise words and even objects, the latter I should imagine being a relatively more severe form than the other.

It may be urged that a similar loss in other senses should be theoretically possible, *i.e.*, that a man could be mind deaf as well as word deaf and unable to understand sounds just as he is unable to interpret words, though he could give evidence (spontaneously only) of hearing them. Or even one might suppose either smell or touch to be similarly affected. But apart from the extreme difficulty of proving such defects, vision is in man undoubtedly the highest intellectual sense, and probably on that account its acquirements are the easiest lost or damaged. This may be stated in other words, that of all the senses in man, I should be inclined to say—smell ranking next to it in this respect—it is the one least dependent on the others for association, that whereas the stimulus of, and *ipso facto*, the memory of the impression of hearing, touch, &c., are associated usually with those of others, that of vision is often quite solitary, and therefore if lost would have fewer aids to revivification, re-ideation and memory. A sound we hear is generally associated with the sight, perhaps too the tactile perception of its causal agent, a touch equally with vision, sometimes with hearing or even other sensations, while a visual impression may arise *per se* quite unaccompanied by any other sensory stimulus, and so be much more liable to loss of representation, having no other associated memory that it is possible to recall.

It may be here mentioned that a possible explanation of the angular gyrus being particularly liable to lesions and partial lesions lies in the fact of its being at the junction of the different blood supplies, the posterior and middle cerebral, and though usually being supplied by the latter, the former occasionally instead or in addition giving blood to it.

It would seem curious that no analogous condition to either word or mind blindness should have been ever described as resulting from occipital lesions, but I would venture to submit again that this coincides with the theory enunciated in this paper, the angular gyrus being peculiarly for distinct central vision as used when the object is to be and is most clearly defined, and *ipso facto* most clearly perceived, and a "visual impression" *sic* of it retained and rendered capable of future representation, while the occipital lobe is solely for general binocular vision; possibly every portion of this relatively large area being divided into functionally separated segments related to correlated separated portions of the retina and the general visual field, even without supposing these to be a separate portion of it required for the representation of colour vision; for it is well known that cases have been recorded of hemiopia for colours alone, there being now a good many recorded, and from this fact several authorities have considered a separate cortical centre for colour vision to be necessarily present.

The affection has been usually attributed to lesions of the occipital lobe. I am not aware of any case having been found due to disease of any part of the parietal lobe; indeed in the cases of amblyopia the colour fields are generally reduced in ratio with that for light, as is the ordinary rule in hemianopsia of cortical origin, and in chiasmatic (temporal hemiopia) the colour fields are equally lost. In optic nerve atrophy, as is well known, colour blindness is in nearly all cases well marked, and seems often at least to bear no relation to the loss of visual acuteness.

It may possibly be that there is a separate centre for colour vision in the cortex, and if so, colours not requiring fixed and clearest vision for their perception, it would presumably be in the occipital lobe. But if the physical perception of colours really depends for each one on the relative number of vibrations of light ether, it may also well be that the same part of the cortex as for light subserves this formation, and that a less severe lesion, *i.e.*, a relative one, could cause this symptom, it may possibly have some relation to the function of different layers of cortical cells. I am not aware if it has ever been shown to follow lesion of the tract or radiation which would, of course, under the theory of a separate area for its centre, be quite possible; also it seems that in most cases of "hemiachromatopsia," there is generally some affection of ordinary vision, and that as the loss for each colour is often so very different, and that it is possible for persistent blindness to one or more colours to remain as in the case quoted by Gowers ("Manual," vol. ii., p. 146), while the others may be imperfectly perceived, it would appear to be necessary, if there is a separate area for the centre for colour vision, that there should be one for each individually and separately. It is much more reasonable on these grounds to suppose the colour loss to be due to a lessening of function, which, if more pronounced would cause a loss for light.

I have purposely refrained from alluding to the result of experiments on animals other than monkeys, for in these former the brain is so unlike in general appearance that of the human subject, that it seems as though the clearest evidence for a corresponding nomenclature of parts is a corresponding response to experimental lesion or irritation, and to argue from this backwards as to the seat of a function in similar named areas would, I think, be rather a *petitio principii*, but still speaking broadly and without going into any minutiæ, it seems as though even in these animals there was as in man, a distinct and separate representation for (1) clear vision and (2) for the correlated retinal half fields.

Bechterew (quoted by Ferrier "Cerebral Localisation," p. 65) found in dogs and cats two regions functionally differentiated in this way, one posterior related to the retinal halves, and another anterior to this in the middle of the second external convolution in relation with the opposite eye alone. It seems as though the same facts may be deduced from the very opposing results recorded by Dalton, Loeb, Goltz, Luciani, Tamburini and others, some stating that lesions of the visual areas in these animals caused crossed amblyopia, while others declared the visual loss to be a hemiopic one.

I cannot help thinking that the explanation is such as has been stated; and that it will be found on examination that the general scheme of visual representation in nature is for the whole field of the opposite eye most probably as in man, each eye being represented on both sides of the brain, though this is impossible to determine in animals; and that in those animals provided with binocular vision the homologue of an occipital lobe exists for correlated halves of each retina.

THE SENSORY SIDE OF APHASIA.[1]

BY E. A. SHAW, B.A., M.B., SURGEON, R.N.

THE consideration of this subject may best be commenced by a short sketch of its history, which is indissolubly welded with that of the localisation of the faculty of language as a whole in the brain cortex. Gall appears to have been the first to have placed the faculty of speech in the anterior lobes of the brain, regarding it as more or less in relationship with the frontal eminences of the skull, but it would seem that he so placed it from *a priori* reasoning, and more as a quasi-scientific speculation than as an induction from the consideration of an assemblage of ascertained facts. The first serious attempt to locate it by means of morbid anatomy seems to have been made by Bouillaud in 1825, who, in his "Traité de l'encéphalite," placed it in the anterior lobes of the cerebrum.

The next worker on the subject appears to have been Marc Dax who is stated to have published at Montpellier in 1836 an account of a "Lésion de la moitié gauche de l'encéphale, coïncidant avec l'oubli des signes de la pensée," in which he localized it in the left hemisphere near to the island of Reil. Bernard, however, in his work on "l'Aphasie" published in 1884, goes very deeply into the question of the validity of the claim of Marc Dax to be regarded as almost the pioneer in this field of pathology, and the evidence he adduces makes it very doubtful whether Dax's memoir, though doubtless it existed in 1836, was ever read publicly; it was apparently first published only on the 25th May, 1865; and was certainly all but forgotten till Broca's famous and now historical paper " Sur le siège de la faculté du langage articulé avec deux observations d'aphémie," appeared in the *Bulletin de la Société Anatomique*, tome vi., August, 1861, and placed the faculty of language in the third left frontal convolution.

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[1] Communicated by the Director General of the Medical Department of the Navy.

In the same year Charcot published in the *Proceedings of the Anatomical Society of Paris* a most interesting case (quoted at greater length in the list of cases) in which there was right hemiplegia and aphasia, and at the *post-mortem* the frontal part of the brain was found to be perfectly normal, but the first and second tempero-sphenoidal convolutions, island of Reil, extra-ventricular nucleus of the corpus striatum, and intra-ventricular nucleus in its posterior half had been destroyed by softening. Broca appears to have examined the case during life, and was certainly present at the *post-mortem,* which is described as having been conducted with the greatest possible care, and at which he admitted that this case was distinctly at variance with his hypothesis. Its true bearing we now easily see, but for some time it was regarded as an insuperable objection to Broca's hypothesis, and then later, as cases similar to Broca's multiplied, as one of those exceptions that prove the rule.

Montpellier seems to have been peculiarly connected with the subject of aphasia, for one of its professors, M. Lordat, presented a most striking instance of the disease, viz., entire inability to speak, due to loss of memory for words with complete word-deafness and word-blindness : he could spell but not read ; in his own words "Je possédais complètement la partie interne du langage, je rien avais perdu que la partie externe."

Hughlings Jackson (*London Hospital Reports,* 1864) first brought into prominence the distinction between the use of emotional speech (oaths, interjections, &c.) and conceptional or propositional speech (*i.e.*, speech embodying some idea).

Marce (*Mem. de la Soc. de Biol.*, tome iii., 1865) was the first to maintain that the centres of co-ordination for spoken and written words are different and separated from each other.

Ogle (*St. George's Hospital Reports*, 1867) arrived at a similar conclusion from a consideration of cases in which agraphia and aphasia were separated; he also placed the two centres close together, and drew a distinction between "amnemonic aphasia" in which inability to translate ideas into symbols was due to forgetfulness of words, and "atactic aphasia" in which the defect was due to want of cöordinating power over the muscles of articulation; and further applied the same distinction to agraphia.

Jaccoud (*Leçons de clinique médicale*, 1874, page 65) supposed automatic speech to be presided over by a basal centre of sound situated underneath the corpora quadrigemina, but this was not much more than a speculation.

Charlton Bastian, according to a review by de Watteville in BRAIN for July, 1885, appears to have been the first to draw attention to the sensory nature of word-blindness and word-deafness in an article "On various forms of Loss of Speech in Cerebral Disease," which appeared in the *British and Foreign Medical Chirurgical Review* for 1869. Broadbent, in a remarkable paper on the "Central Mechanism of Speech and Thought" (*Medico-Chirurgical Transactions*, 1872), adopted the hypothesis of distinct perceptive centres; and Wernicke ("Der Aphasische Symptomen-Complex," Breslau, 1874) still further defined the localisation of an auditory word-centre and placed it in the first temporal convolution. And since this the subject has received much attention at the hands of Bastian, Broadbent, Kussmaul, Habershon, Wernicke, Kahler and Pick, Fritsch, Ross and Giampietro.

I now propose to quote a number of cases which I have collected from various sources, including one of my own. All these have been selected from the very extensive literature of the subject as illustrative of the sensory side of aphasia, and in addition as having a *post-mortem* record attached; they will be followed by a tabular statement of their main features, both clinical and pathological.

Case 1.—The first case of importance published after Broca's celebrated paper was the one by Charcot, which appeared in the *Proceedings of the Anatomical Society of Paris*, late in the year 1861. The patient was a woman of 47, who, consequent on a fit of apoplexy, presented right hemiplegia and aphasia. Her intelligence was unaffected, and memory good, but her articulate language was reduced to the monosyllable "Ta" repeated several times over ("ta, ta, ta, ta") very rapidly and distinctly each time she tried to answer any question or to communicate her own ideas. The tongue was perfectly free and could be moved in any direction.

Post-mortem.—It was found that softening had destroyed the first and second temporo-sphenoidal convolutions, island of Reil, extra-ventricular nucleus of the corpus striatum and intra-ventricular nucleus in its posterior half; the optic thalamus was intact; the frontal convolutions presented no alteration either in volume, colour or consistence. The examination is described as having been conducted with the greatest care in the presence of MM. Cornil and Broca, the latter of whom, as has already been mentioned, admitted the case to be at variance with his hypothesis.

Case 2.—A case published by Bateman in his work on "Aphasia." Wm. Sainty, a waterman, aged 51, on Dec. 9, 1864, went into a tavern to order some beer, when he found himself totally unable to speak. There were no other paralytic symptoms; memory and intelligence were unimpaired. At the end of a fortnight he could speak a few words, but in March, 1865, he had a kind of fit, falling down and remaining for a few moments unconscious. He was then admitted into hospital. Intelligence unimpaired, no want of sequence in his thoughts, partial agraphia, can form one or two words but cannot write a complete sentence. Deglutition unaffected, movements of tongue unimpaired, but right half is slightly raised above left half and is more flabby, and when told to protrude it, he holds it out a long time as if from defect of memory, probably not remembering what he had done. Organs of special sense unimpaired. In May, there was a slight improvement more marked in writing than speaking. During 1866 he had fits every few weeks.

Re-admitted January 12, 1867. No diminution of intelligence or motor power, no paralysis. Understands all that is said, but has lost the memory of words so far as substantives are concerned. If asked to bring anything he brings the right article, but if he wishes to ask for anything himself, he usually asks for the wrong thing, afterwards correcting himself, saying that he knew it but could not recall its name, and on being asked was it this, that or the other thing, he invariably recognised the proper name for it.

April 24, 1867.—Sudden lower paraplegia and speechlessness, upper limbs unaffected. In May he was admitted into the Borough Asylum, quite imbecile and unmanageable.

July, 1868.—Though unable to articulate he gesticulates frightfully, endeavouring by language of signs to supply the loss of articulate language. He died at the end of the month.

Post-mortem.—Intense congestion of convex surface of hemispheres especially on left side, considerable opacity of arachnoid also on left side, a *little* flattening of convex surface of left anterior lobe. In middle of left posterior lobe was a softening about the size of an apricot; a similar condition existed at the same spot in the right hemisphere, but the softening was not so far advanced. The frontal convolutions were carefully examined, as were the central ganglia, pons and cerebellum, and all found healthy. These points were subsequently verified by microscopical examination.

Case 3.—Also reported by Bateman in the same work. Mr. N., a merchant, aged 51, consulted him in March, 1867. He was able to speak, but on being questioned got confused, saying he could not find words to express his symptoms; what answers he made were quite coherent, but given in the fewest possible words. He had lost the memory of many substantives and would call things by their wrong names, afterwards correcting himself.

Post-mortem.—At point of union of middle one-third with posterior one-third of convex surface of left hemisphere was an arachnoid cyst under which was a well circumscribed portion of softened cerebral tissue about the size and shape of an apricot. Anterior hemisphere was quite healthy.

Case 4.—Published by Broadbent in a paper on the "Central Mechanism of Speech and Thought," *Medico-Chirurgical Transactions*, vol. lv., 1872, p. 162.

An intelligent man of 59, after an acute cerebral attack lost completely the power to read printed or written words; he was also unable to recall the name of the most familiar object presented to him. He could, however, converse fluently, his vocabulary was large, and his words well-chosen and arranged, although he occasionally forgot the names of streets, persons and things. He could also write easily and correctly, both from dictation and spontaneously.

Post-mortem.—He died from an extensive hæmorrhage into the left temporal lobe with rupture into the lateral ventricle. Two foci of softening of older date were observed, one being situated in the temporo-sphenoidal lobe beneath the posterior end of the parallel sulcus, the other higher up underlying the angular gyrus, and between it and the point where the descending horn of the ventricle is given off.

Case 5.—Also published by Broadbent in the *Medico-Chirurgical Transactions* for 1878.

A man aged 60, who had previously been a good talker and a great reader, suffered, after a fit of some sort, from a peculiar affection of the speech, with paresis of the right side of the face but no hemiplegia. His speech was a mere inarticulate jargon. When asked a question he would make a brief reply as if he understood and answered; the modulation of the voice and the emphasis were perfectly natural, and corresponded with the facial expression and gestures, but as a rule there was not the least semblance to words in what he said. His replies were often so suitable in length and emphasis that it might have been supposed that he had comprehended the question. When, however, he was told to do anything it was seen that he did not understand the simplest phrase. He sat up in bed once or twice when required to do so, but as this was not made a test question, there would be other indications of what was wanted, and he was extremely ready in comprehending signs. When told to give his hand he invariably put out the tongue. A letter addressed to him at the hospital being handed to him, he took it, appeared to read the name and address and put it down again, not attempting to open it. A piece of paper having on it, " Give me your hand" was handed to him; he took it, held it so as to get a good light on it, then having apparently read it, laid it aside without giving his hand, though asked to do so by word of mouth as well as in writing.

Autopsy.—A large focus of softening was found in the left hemisphere limited to its posterior half. Part of the supramarginal lobule was yellow in colour, shrunken in volume and soft. This condition extended upwards and backwards to within about half an inch of the longitudinal fissure just in front of the external parieto-occipital fissure, involving therefore the postero-parietal lobule. The morbid change implicated the angular gyrus and nearly reached the occipital lobe. The adjacent parts of the temporo-sphenoidal lobe, the posterior end of the infra-marginal and the parallel gyri were soft but not wasted or discoloured on the surface.

Case 6.—A case published by Kahler and Pick " Beitrag zur Lehre von der Localisation der Hirnfunctionen," *Prager Viertel Jahrschrift*, bd. cxli., 1879. There was well marked word-deafness, and *post-mortem* there was found a lesion situate in the first and second temporo-sphenoidal convolutions of the left side.

Case 7. — A similar case published by Fritsch, *Wiener Medizinische Presse*, 1880. Well marked word-deafness, and *post-*

mortem a lesion of first and second temporo-sphenoidal convolutions of left side.

Case 8.—Also a case of word-deafness published by Wehrnich, "Lehrbuch der Gehirnkrankheiten fur Aerzte und Studirende," Band ii., 1881. At the autopsy a focus of softening was found in the first and posterior part of the second temporo-sphenoidal convolutions of left side.

Case 9.—A case of severe amnesic aphasia reported by Kussmaul in his article on "Disturbances of Speech" in Ziemssen's Encyclopædia.

A stonemason of 55, in the clinic at Freibourg, presented a typical picture of loss of memory for spoken and written words without special complications, this manifesting itself mainly by a forgetfulness of substantives and the markedly disjointed manner in which he combined letters and syllables in spoken words; recognition and comprehension of words not being noticeably affected.

No note about vision save that there was much presbyopia.

Autopsy.—Two foci of yellowish-red circumscribed softening on left side; one 5.5 cm. x 1.1 to 1.8 cm. occupied the anterior position of the gyrus angularis; the other 2 cm. x 0.6 cm. had destroyed partially gyrus occipitalis ii. where it unites with gyrus temporalis ii.; the depth of these foci was only 4 mm.

Case 10.—A case reported by Broadbent "On a particular form of Amnesia; loss of nouns," *Medico-Chirurgical Transactions* for 1884.

A gentleman of 72, developed slight hemiplegia in September, 1878, and died in December, 1883. All this time he seldom used a noun-substantive, and when he did, it was wrongly used. He could not name an object at sight, could not repeat nouns after hearing them, though when told to say "1, 2, 3," did so promptly. He clearly understood all that was said to him, read newspapers and pointed out paragraphs relating to people he knew. His memory was good, but he could not answer a question in writing. There was well-marked right hemiplegia and anæsthesia.

Autopsy.—The right hemisphere was normal.

Left Hemisphere.—Posterior *outer* and two long posterior convolutions of the island of Reil had entirely disappeared. The angular gyrus round the margin of the Sylvian fissure and the super-marginal gyrus forming its upper margin posteriorly were undermined by an extensive area of degeneration continuous with that which has destroyed the posterior insular convolutions. The auditory perceptive centre had entirely escaped. The mass of fibres running from behind forwards under posterior convolutions of island of Reil, converging from angular, supra and infra-marginal, parallel and other gyri to the two ascending parietals and posterior part of third frontal, had been destroyed.

Case 11.—A case reported by Osler in the *International Journal of Medical Science* for March, 1891.

J. W., aged 72, a Scotchman, by occupation a book-keeper. Inability to read a newspaper was the first sign; there was typical word-blindness. Philadelphia he spelt aloud "Pringrek." He named objects held before him quite readily; there was no word-deafness at all. There was misplacing of words and sentences (paraphasia). In alluding to a wetting, he described it as "deliberate attacks of wet dress." There was right lateral homonymous hemianopsia, no paralysis. These conditions persisted over two months with gradual loss of muscular strength and mental power. For thirty-six hours before death there was paralysis of right arm and leg.

Autopsy.—Neurotic softening in the left hemisphere of the supra-marginal and lower part of the angular gyri, of the posterior part of the first and second temporal and of the two annectant convolutions uniting the first temporal to the parietal; there was complete transverse softening of the white matter between these convolutions externally and the lateral ventricle; the grey and white matter of the occipital lobe was uninvolved, but there was interruption of the fibres of the optic radiation. The anterior lobes and right side of hemispheres were healthy.

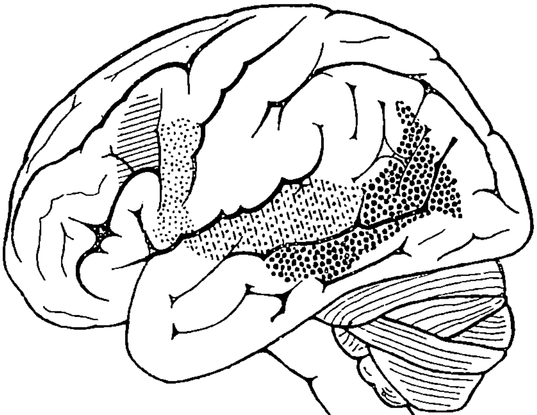
Case 12.—A case reported by myself in the *British Medical Journal* for February 27, 1892.

M. A. W., aged 72, widow, housekeeper, admitted into West Riding Asylum, January 30, 1890. She exhibited almost complete aphasia, agraphia and considerable dementia. She persistently repeated "Win yo? win yo?" (will you) and "man," and these were the few intelligible among many absolutely unintelligible utterances. She would frequently attempt to write, but would allow the pencil to fall from her fingers and go on with the motions of writing as though it was still there; she never succeeded in making a single recognisable letter. She was quite cheerful and accompanied her attempts at talking by gestures, the meaning of which was quite obscure, hence it could not be distinctly ascertained whether word-blindness as well as word-deafness was present. There was some paralysis of right side of face, not absolute;

distinct feebleness of right lower limb shown in walking but no dragging. Right upper limb was paralysed for fine movements, but was free from contractions and retained moderate grasping power.

February 26.—She could not apparently grasp the meaning of the most simple remark supplemented by gestures which would ordinarily make the meaning quite unequivocal. For months before her death in September, 1891, she made repeatedly most vigorous attempts to make herself understood, getting quite vehement at times but only succeeding in uttering unintelligible jargon, and often weeping at her consequent failure to make herself understood.

Post-mortem.—Diploë of skull cap softened. Dura mater a little thickened; there was a considerable quantity of fluid in subarachnoid space, especially on left side, where the visceral arachnoid was raised by it from surface of convolutions and pia mater in some places to extent of half an inch. On left side of brain there was considerable orange-red softening of superior and part of middle temporo-sphenoidal convolutions, and of the angular gyrus and also of the convolution of the surface of the island of Reil. There was a less degree of softening at the junction of the second frontal with the ascending frontal, and the latter itself was affected with softening but to a slighter extent than any of the other diseased areas. On section the white matter between the claustrum and external cortex was seen to be involved also.



ANALYSIS OF CASES.

No. of Case.	*Prominent Clinical Features.*				*Main Pathological Lesions Affecting*							
1	Word-deafness	Speech reduced to "ta-ta-ta"			1st and 2nd temporo-sphenoidal convolutions						Island of Reil	
2			Loss of memory for substantives									Middle of left posterior lobe
3			Loss of memory for substantives									Junction of middle and posterior thirds of convex surface of left hemisphere
4				Word-blindness	Temporo-sphenoidal convolutions	Area underlying the angular gyrus						
5	Word-deafness	Speech jargon		Word-blindness	Temporo-sphenoidal	Angular gyrus	Supra-marginal	Postero-parietal				
6	Word-deafness				Temporo-sphenoidal I. and II.							
7	Word-deafness				Temporo-sphenoidal I. and II.							
8	Word-deafness				Temporo-sphenoidal I. and II.							
9		Propositional defect in speech	Loss of memory for substantives			Angular gyrus			Occipital II.			
10			Loss of memory for substantives			Angular gyrus	Supra-marginal			Commisural fibres from hind- to fore-brain	Island of Reil	
11			Propositional defect	Word-blindness	Temporal I. and II.	Angular gyrus	Supra-marginal					
12	Word-deafness	Speech jargon			Temporo-sphenoidal I. and II.	Angular gyrus					Island of Reil	

Proceeding now to a consideration of these cases, let us see what may be deduced from them. I am only too conscious that they are lamentably few in number for the purpose, the cause of this being the limitation of cases chosen to those with a *post-mortem* record attached, and in the most exhaustive possible examination of the literature of the subject, though I have met with some hundreds of reported cases of sensory aphasia, these twelve were the only ones I found with such a record.

Taking first those presenting the features of "word-deafness" we have six cases, Nos. 1, 5, 6, 7, 8, and 12, and each of these cases is associated with disease of the temporo-sphenoidal convolutions; in five of them the convolutions are distinctly specified as being the first and second, in the remaining one (case 5) it is simply stated that part of the temporo-sphenoidal lobule was softened. The association is obvious. On the other hand, we have two cases, Nos. 4 and 11, in which there was disease of this region without apparently any word-deafness at all. Of course it is possible that the lesion was not sufficiently severe to produce this effect in these two cases, and we may regard this as neutral evidence so far as the localisation of a cortical centre associated with word-deafness is concerned, for the series does not contain a single case exhibiting word-deafness in which there was not disease of the temporo-sphenoidal convolutions with the one doubtful exception of case 10, in which the patient was unable to repeat nouns after hearing them, and this Broadbent regards as a case not of word-deafness, but of loss of memory for substantives, of which the series I have collected presents several instances. Therefore, from the pathological standpoint we may say there is a distinct association between the first and second tempero-sphenoidal convolutions and the phenomenon of word-deafness; in other words, that in these convolutions is situated the "auditory perceptive centre." This is where Ferrier, from considerations of quite another nature, places it, and though the experimental researches of Schäfer and Sanger Brown as to the position of the auditory centre in monkeys do not support this conclusion, neither do they contradict it; they are of a negative nature. Giampietro (*Ann. des Mal de l'Oreille, &c.*, March, 1893), I see, places what he terms the "ideo-phonic" centre in the first temporal (tempero-sphenoidal) convolution alone.

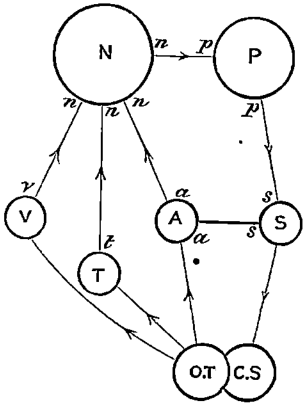
Taking next those cases presenting the features of "word-blindness," we have three cases, Nos. 4, 5, and 11, all associated with disease of the angular gyrus; it is true that in two of these (cases 5 and 11) there is also disease of the supra-marginal convolution, but, for reasons which I shall come to shortly, it is quite unlikely that the supra-marginal convolution has anything to do with word-blindness.

On the other hand, there are two cases (9 and 12) in which there was disease of the angular gyrus, but in which word-blindness is not recorded, but here, as in the consideration of word-deafness one must regard the evidence as negative, the word-blindness might have existed undetected, or the lesion of the angular gyrus might have been insufficient to have caused it, and as against it we have the fact that no case of word-blindness has been recorded where, *post-mortem*, the angular gyrus has been found free from disease. The location of a cortical centre for vision from considerations of experimental physiology is at present in a vague and unsatisfactory condition. Ferrier originally placed it in the angular gyrus, but later Munk showed that the occipital convolutions were concerned, and Schäfer went a step further and maintained that the angular gyrus had nothing to do with vision, and that the occipital gyri had everything to do with it, and that cases in which blindness was associated with disease of the angular gyrus, were simply due to the fact that the fibres connecting the optic tracts and the occipital convolutions, which run under the angular gyrus, had been destroyed. However, the three cases which have been adduced must be regarded I think, so far as they have any value at all, as supporting the view that the angular gyrus has a connection with what might be called "word-vision" rather than the occipital gyri, for in not one of them is the occipital lobe mentioned as being diseased; and also that there is a special centre for word-vision, rather than that word-vision is the special activity of the general visual centre which is the first to be lost on its being affected by disease.

And now I come to the consideration of a variety of sensory aphasia, about which there has been much discussion, the very existence of which is denied by some writers, viewed as doubtful by others.

Broadbent is its most powerful advocate, and he names it "loss of nouns" or "loss of memory for substantives," regarding case 10 as a typical instance of it; and it may be noticed that in three other of the cases I have collected (Nos. 2, 3, and 9), it is mentioned as a particular feature.

Broadbent's diagram is as follows (BRAIN, vol. i., p. 493, 1878):—



V.—Visual centre (angular gyrus).
T.—Tactual „ (uncinate gyrus).
A.—Auditory „ (infra-marginal gyrus).

All sending tracts of fibres as indicated to "*N*" *the naming centre.* Here preceptions from V and T are combined into an idea, which can be symbolised by a name reaching N through A which has always been in the experience of the individual associated with the idea or object.

P.—Propositional centre in which a phrase is formed and mentally rehearsed.

S.—Motor speech centre.

Lesion of V or fibres V to N causes word-blindness.

„ A or fibres A to N causes word-deafness; and mistakes in words are not recognisable by patient, and A being the cortical guiding centre for S, lesion of fibres between A and S may result in mistakes in words which however, are recognisable by patient.

Lesion of S causes ataxic aphasia.

„ N would cause loss of memory of names or nouns.

„ P would leave patient unable to contruct a sentence, though retaining use of nouns.

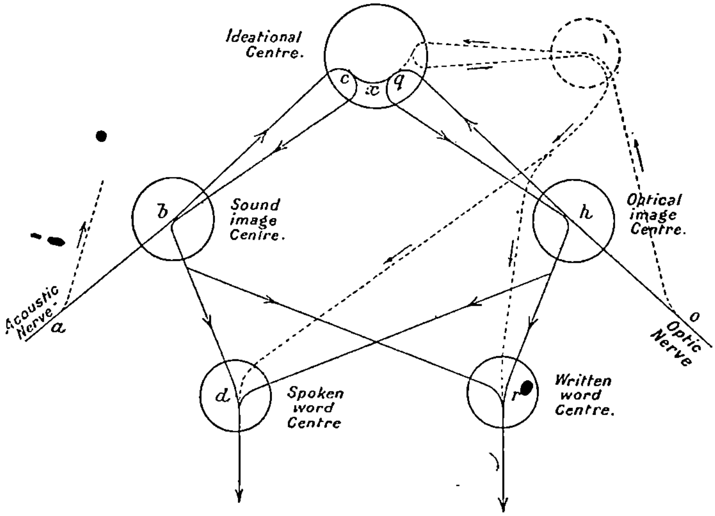
Broadbent, in Brain, vol. i., 1878-79, p. 491 *et seq.*, puts forward the idea of a motor cell group—a "word group" situated probably in the corpus striatum— and postulates a distinct "naming" centre and also a "propositional" centre, in which phrases are formed and mentally rehearsed; the existence of this "naming centre" he further dwells on in an article in the *Medico-Chirurgical Transactions* for 1884, "On a Particular Form of Amnesia," regarding the case therein reported (above quoted as case No. 10) as due to the destruction of fibres connecting the "naming centre" with the motor-speech centre (third frontal convol.). Charlton Bastian (*British Medical Journal*, October 29, 1887) regards this propositional centre as merely a special activity of the general function of language, and the "loss of nouns" as a mere upsetting of the whole structure of language, and therein states that Ross holds similar views, though the latter in his work on "Nervous Diseases" ("Aphasia" in vol. ii.) mentions "Loss of Memory of Names or Nouns" as a subdivision of amnesic aphasia. Bastian further maintains (*Clinical Journal*, May 17, 1893) that "in ordinary silent thought the revival of words occurs primarily and perhaps mainly in the auditory centre."

There is, of course, in the very nature of the subject, any amount of room for speculation, and the thought naturally suggests itself, why should the revival of words in thought occur in the auditory rather than in the visual perceptive centre, to which it has been replied that the storing up of words in the memory is commenced and carried on for years as the child grows up, through the sense of hearing, and that from time immemorial in the majority of instances this has been the only way in which they have been so stored, the addition of words to the memory through the visual sight centre in reading, in both the history of the race and the individual, having been a recent and subordinate process assisted or possibly mainly carried out in most cases by the sense of hearing in the act of reading aloud, which is a not altogether improbable explanation why in the majority of educated people, new words and ideas are so much more easily grasped by means of lectures than by reading. Though the subject is, as yet, one mainly of conjecture, an examination of the schemes that have been suggested in explanation of the phenomena of voluntary speech may not be out of place.

There is no positive evidence of the existence of a naming centre; we have simply the occurrence of cases in which there is the one simple defect, the "loss of nouns"; and Broadbent says he has no evidence of a propositionary, as apart from a speech, centre, beyond the fact that cases are met with in which the patient cannot frame the simplest phrase and yet can repeat anything; these he would explain by assuming the propositional centre to be destroyed, the speech centre being intact. The converse, he says, would be conclusive, *i.e.*, the existence of cases in which the power of speech was lost, while the integrity of the propositional centre, as shown by the expression of ideas in writing, remained intact. I have come across three such cases, one reported by Trousseau (*Clinique Méd.*, tome ii.), another by Bouillaud (*Bull. de l'Acad. Méd.*, tome xxx.), and the third by Boinet (*Gaz. des Hopît.*, tome iii.); in all three patients the power of speech was lost, but their intelligence was unaffected, and all operations were perfectly conducted by means of writing. These certainly are cases for consideration which lend no little support to the view of the existence of a propositional centre, though why this centre should not be able at the same time to carry on the duties of the "naming" centre is a little difficult to see, and Kussmaul appears to be of this opinion, for in his scheme he regards them as forming one centre, which he terms the "ideational" centre.

Kussmaul's scheme is one which certainly cannot be accused of lacking completeness.

Comparing this scheme with Broadbent's, it will be seen that they have many points in common, the main points of difference being the fusion of Broadbent's "naming" and "propositionary" centres into one, the "ideational" centre of Kussmaul; the separation of Broadbent's speech centre into the two, one for spoken the other for written words, of Kussmaul; Broadbent, though he says (BRAIN, vol. i., 1878, p. 493) that the auditory centre is the cortical guiding centre for speech, does not represent the path from his propositionary centre to his speech centre as passing through the auditory centre; this omission is rectified in Kussmaul's schema. With regard to the path "o p d," which the latter regards as the one travelled in reading off aloud an uncomprehended written word, I would suggest that considering the close relationship which has always existed between the sound image centre and the spoken word centre in the history both of the individual and the race; the much remoter con-



Kussmaul's Scheme.

a, b, c, b, d.—Collective acoustic motor track for spoken speech.

o, p, q, p. r.— ,, optic ,, ,, written ,,

Punctated lines indicate paths from nerves of sense through other image centres to centre for conceptions (melodies, songs, gestures, &c.)

a, b, d.—Track for repetition of uncomprehended words.

o, p, r.— ,, copying ,, ,, ,,

c, b, d.— ,, conceptions expressed in spoken words.

q, p, r.— ,, ,, ,, ,, written ,,

c, x, q.— ,, of connection between sound and text images, and renders feasible the change of spoken into written symbols through medium of thoughts.

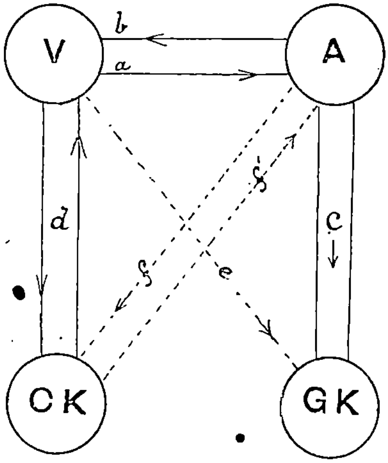
a, b, r.—Track for writing down of an uncomprehended word that has been heard.

o, p, d.—Track for reading off aloud an uncomprehended written word.

a, b, c, b, r.—Writing down dictated words that are understood.

o, p, q, p, d.—Reading aloud written words that are understood.

nection, and, if it exist, quite recently developed one as compared with the other, between the optical image centre and the spoken word centre; and also the fact that in a case of word-deafness, a patient, though he might make an attempt to read off aloud an uncomprehended word, would be quite unable to tell whether or not he was making a good or bad attempt; in short, regarding the auditory centre as the guiding centre for speech, I would suggest that the path "o p d" is hardly likely to be the correct one for the reading off aloud of an uncomprehended word, and that the more probable condition is the existence of a path from the optical image centre to the sound image centre, and that in the above instance the track is along this path and so to the spoken word centre;



Bastian's Scheme.

V.—Visual word centre.
A.—Auditory ,, ,,
C K.—Cheiro-kinæsthetic word centre (writing).
G K.—Glosso-kinæsthetic word centre (speech).
d.—Visuo-kinæsthetic commissure.
b.—Audito-visual ,,
a.—Visuo-auditory ,,
c.—Audito-kinæsthetic ,,
f.—Audito-cheiro-kinæsthetic commissure
f'.—Cheiro-kinæsthetic-auditory ,,
e.—Visuo-glosso-kinæsthetic ,,

} unimportant, stimuli only seem to pass along under "exceptional circumstances," which are not discussed.

and applying a similar line of argument, *mutatis mutandis* to the path "a b r" for the writing down of an uncomprehended word that has been heard, I should prefer to assume the existence of a path from auditory to visual centre and so to the written word centre rather than direct from auditory to written word centre.

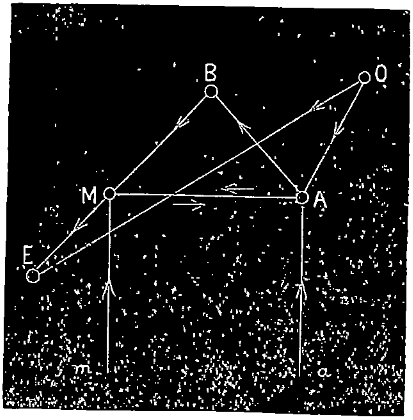
The existence of this cross-communication both ways between the auditory and visual centres is assumed by Charlton Bastian in his schema (*Brit. Med. Journal,* Oct. 29, 1887) which I shall now consider.

It will be seen that Bastian has no centre corresponding with either Broadbent's "naming" or "propositionary" centre or with Kussmaul's "ideational" centre; the functions of such possible centres he apparently relegates to the auditory centre; his own words are (*Clin. Journal,* May 17, 1893): "In ordinary silent thought the revival of words occurs primarily and perhaps mainly in the auditory centre. As I am speaking now, they come up quasi-spontaneously in the auditory centre"; and again (*Ibid.*): "In the same way, in the case of a person writing, the visual centre would be roused either primarily from the auditory centre, or, as in copying, by stronger impressions coming from without." Why the auditory centre should have been specially selected as the seat of that function of mind controlling thought does not seem quite clear, neither, assuming for a moment the truth of this hypothesis, does the course of the path by which thoughts are called up in the auditory centre by sensations of touch or taste, nor yet why thoughts thus called up should be developed in the auditory centre rather than elsewhere.

I now come to a consideration of Lichtheim's views, as propounded by him in a translation of his manuscript article by de Watteville in BRAIN, January, 1885; which appeared after being carefully revised by himself. Therein he puts forward several alternative schemes, the one receiving on the whole his preference being the one marked A on the next page.

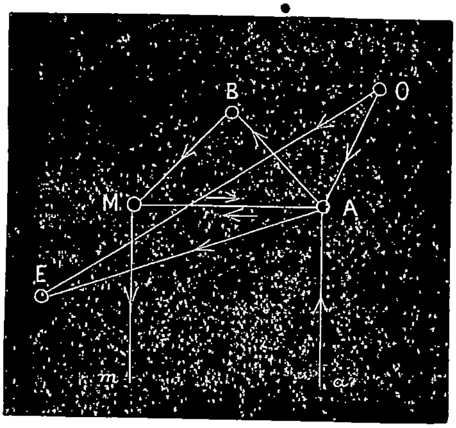
Having, however, a certain amount of doubt as to whether B M E be the path for volitional writing, he suggests as an alternative the diagram marked B.

It will be seen that in the latter diagram he does away with the direct communication from M to E, and makes his path for volitional writing B M A E.



(A)—*Lichtheim's Scheme.*

A.—Centre for auditory word impressions.
M.—Centre (kinæsthetic) spoken word impressions.
B.—Conceptional Centre.
O.—Visual word impressions.
E.—Written word impressions.
Reading aloud—O A M m. (The arrow should point downwards.)

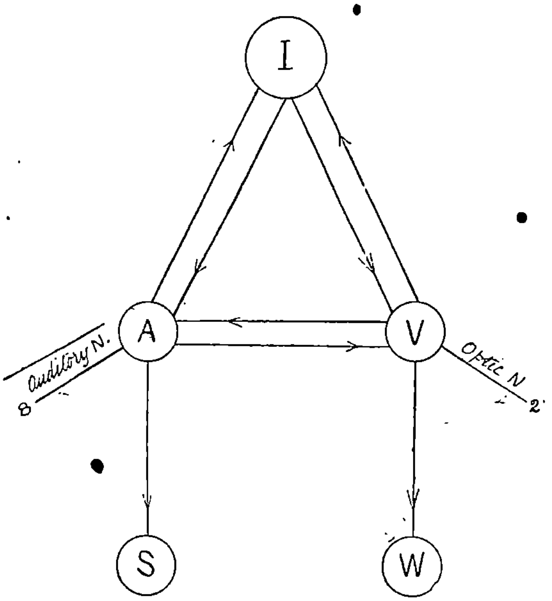


(B)—*Lichtheim's Alternative Scheme.*

It is hardly to be supposed that Lichtheim would deny the existence of a path of communication from O, the sensorial visual centre, to B, the concept centre, though his diagram does not recognise its existence, and presumably, confining one's self to the question of language alone, the path by which words in reading would reach B would be through O A B. Indeed, the Berne professor regards the auditory word impression centre as of primary importance; he says (*Ibid.*, p. 439) that damage to it involves the loss of understanding of written language (?) and as his diagrams show, the visual word impression centre he regards as communicating with the conceptional centre only through A. Now in the early stage of a previous education, this, I take it, undoubtedly is the case; the individual has to assist the process of grasping an idea from written symbols by reading aloud, and at a somewhat later stage, though the reading aloud is dispensed with, one sees the lips moving in correspondence with the words read; but in an advanced stage of education I think there is good reason for assuming the existence of a direct communication, recently established no doubt, between the visual word impression centre and the concept centre.

His views on the subject of word-blindness are somewhat at variance with those of other writers on the subject. He states (*Ibid.*, p. 484) that in cases of isolated word-blindness the patients can copy, and that in such cases we have not to do with a focal lesion in O, and that the break is in the tract O A, and thus again the visual word centre is subordinated to the auditory word centre. But surely it is possible by an injury to the visual word centre for word-blindness to be produced, and if A really occupies the position of all primary importance assigned to it by Lichtheim, how is a man who has lost the communication between it and O to copy words? The whole case for this assumption, it appears to me, depends on the statement on page 439, that damage to A involves loss of understanding of written language, which, though it may be the case in imperfectly educated persons, is not so, I venture to think, in their intellectual superiors, and hence a scheme providing for both possibilities becomes necessary.

I would now venture to suggest, not without considerable diffidence, the following as a scheme, including as far as possible the good points of its predecessors, at the same time minimising their defective ones, and also embodying the present state of our knowledge of the subject as far as may be done. Of course it is bound to have defects and is largely speculative, but I venture to think it will serve as a working hypothesis for the explanation of by far the great majority of cases of speech defect.



I.—Ideational centre, naming, propositionary, can change spoken into written symbols "through medium of thoughts."
A.—Auditory centre, 1st and 2nd temporo-sphenoidal convolutions.
V.—Visual centre. Angular gyrus.
S.—Spoken word centre, 3rd left frontal convolution.
W.—Written word centre, 2nd left frontal convolution.
8, A, I, A, S.—Collective acoustic motor track for spoken speech.
2, V, I, V, W.—Collective optic motor track for written speech.
8, A, S.—Repetition of uncomprehended words.
2, V, W.—Copying of uncomprehended words.
I, A, S.—Conceptions expressed in spoken words.
I, V, W.—Conceptions expressed in written words.
8, A, V, W.—Writing down of an uncomprehended word that has been heard.
2, V, A, S.—Reading off aloud an uncomprehended written word.
8, A, I, V, W.—Writing down dictated words that are understood.
2, V, I, A, S.—Reading aloud written words that are understood.
Centres of perception for touch, taste, smell, &c., have been left out for sake of clearness.

I have applied it to a large number of the published cases of speech defect of the kind under discussion, and I have in every one yet tried found it capable of affording an explanation, speculative and theoretical though it may be, of all the clinical phenomena recorded.

I now come to a consideration of the nomenclature of this subject.

Ogle gave us the terms "amnemonic aphasia" and "amnemonic agraphia"; Ross speaks of amnesic aphasia, with subdivisions "word-blindness," "word-deafness," and "loss of substantives," as do many others: Broadbent, Bastian, Kussmaul, Lichtheim, &c. Kussmaul speaks of "simple aphasia of recollection." For speech-deafness Lichtheim proposes the word "logokophosis." It is a generally accepted theory that the language of medicine should be as cosmopolitan as possible, and with this object in view, the tendency is to fall back on the Greek language when attempting to coin words to express newly-discovered conditions. I would therefore venture to suggest that the terms "aphasia" and "aphemia" be restricted to defects in the processes of outgoing language of cerebral and bulbar origin respectively; that amnesia be used to mean Kussmaul's "simple aphasia of recollection."

I would suggest the word "aphemæsthesia" to express the general condition of failure of word perception, defect in ingoing language; and for inability to comprehend written symbols (word-blindness), *alexia*, a word used by both Kussmaul and Lichtheim in this sense. For inability to comprehend spoken language (word-deafness); instead of Lichtheim's logokophosis, I would suggest "kophemia," a shorter word whose Greek equivalents κωφος deaf, and φήμη speech, mean precisely the same thing.

Kussmaul's "*paralexia*," in which incorrect words are uttered, is now practically superseded by "*paraphasia*," and rightly, for λεξις means a written, not a spoken word, and the similar defect in writing is equally well expressed by "paragraphia," also a word which may be regarded as now fairly generally accepted.

In conclusion, I would urge that in the recording of these cases, the clinical phenomena should be investigated by means of some such schema as that published by Bastian in his "Paralysis, cerebral, bulbar and spinal," and that the site of any lesion discovered *post-mortem* should be accurately recorded on one of the various brain charts now published for the purpose; and that when cases are published this should appear also; for it is only by the greatest possible accuracy in such details that contradictions in apparently ascertained facts are to be avoided, and a mass of accurate details accumulated, from which the factors governing these phenomena may be safely deduced.

THE "PILOMOTOR" OR "GOOSE-SKIN" REFLEX.

BY JAMES MACKENZIE, M.D.,
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In the careful examination of the skin of patients one frequently comes across a number of phenomena that are extremely difficult to understand. By accurate and minute observation of every detail, light is gradually shed upon the problems involved. They become all the clearer when these phenomena occur in definite areas which one can associate with the distribution of the nerve or blood vessels. When I first found out the occurrence of hyper-æsthesia in visceral disease, it appeared that there was a distinct connection between these areas and the distribution of the spinal nerves. These subjective symptoms sometimes bore a distinct relation in their distribution to those objectively presented in the eruption of herpes zoster. Similar data have been worked up by Dr. Head, into an elaborate delimitation of the fields of cutaneous distribution of the nerves from each spinal segment over the trunk and limbs. The deficiencies in one method of examination were frequently made good by the symptoms present in the other. While the hyper-æsthesia depends for its interpretation on the reliability of the patient, the occurrence of the eruption in herpes zoster, so far as it goes, affords striking and indubitable evidence independent of his sensations. It is very commonly found that patients are extremely inaccurate in regard to their sensations; and one has to be constantly on guard, not only against the patient's misleading statements, but against one's own preconceived ideas. On the other hand, the eruption of herpes zoster is often unreliable on account of the imperfect manner in which one area is affected by the eruption, and also because we can never be certain when an attack of herpes is limited to one nerve root or one spinal segment,—according as we view the situation of the disease. For this reason, studying the subject of the cutaneous distribution of the spinal nerves from the purely clinical aspect, we are left in a somewhat indeterminate position. It behoves us therefore to be on the outlook for any other method that can help to strengthen our position. The results of injuries and lesions of the cord, while affording much valuable evidence, yet often fail us when we desire accurate and minute substantiation, on account of the impossibility of determining the exact limits of the effect of any lesion upon the cord. There are, however, several other phenomena which, if properly understood, can still throw light upon obscure points and substantiate doubtful results obtained in other ways.

The most striking of these is the occurrence of that condition commonly known as goose-flesh or goose-skin (*cutis anserina*). To me it is surprising that a phenomenon so common, and yet so instructive in its manifestations, should have been so universally ignored. All the text books on general medicine, skin diseases and physiology, as well as the larger encyclopædias, either contain no references whatever to this subject, or dismiss it with the briefest notice. Within recent years, two very interesting and instructive articles have been published by Langley and Sherrington,[1] based upon experimental researches, which shed much light on the relationship between the nerves, spinal and sympathetic, and the muscles of the hairs. What goose-skin is does not seem clear. In the New Sydenham Society's Lexicon *cutis anserina* is defined as "a term for that condition of the skin produced by cold and other causes in which the papillæ become rigid and erect, resembling the skin of a plucked goose: goose-skin." According to the same authority goose-skin and goose-flesh are terms applied "to the condition of the skin called *horripilatio*, from its likeness to the skin of a plucked goose." In the same lexicon horripilation is defined as follows: (Latin, *horripilatio*, a bristling of the hair—from *horreo*, to stand on end, *pilus*, a hair . . . .) A sensation of creeping in the skin or as if each hair was stiff and erect in different parts of the body, producing *cutis anserina*." In Billings' "National Medical Dictionary" *cutis anserina* is "goose-skin; condition produced by contraction of pilar muscles over a large area," while horripilation is "shivering, with *cutis anserina*, with bristling of the hair."

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[1] Drs. Langley, J. N., and Sherrington, C. S., "On Pilomotor Nerves," *The Journal of Physiology*, vol. xii., p. 278, 1891; also Langley, J. N., "The arrangement of the Sympathetic Nervous System based chiefly on observations upon Pilomotor Nerves," *ibid.*, vol. xv., p. 176, 1893.

These various definitions are too vague, and they scarcely embrace the full significance of the phenomenon. What all are agreed upon is that the symptom is due to a contraction of the pilar muscle with which each hair follicle is supplied. The symptom, however, need not, and frequently does not, produce a visible erection of the hair; but it usually does cause a projection of the glands or follicles above the skin, giving rise to the peculiar pimply condition that we associate with goose-skin. It also gives rise to that peculiar sensation which is associated with the production of goose-skin. This sensation is peculiar to itself, and to what it is entirely due I am not certain. The mere traction and shrinking of the skin does not appear to be its sole cause, because I find it present in areas where there is no evident goose-skin produced; and it even extends to areas where the hair follicles do not exist. Again, the goose-skin may be present when no such sensation exists. So far as my inquiry into the subject has extended, I find that I am far from having exhausted the information which is to be obtained from the study of this symptom. While it is capable of production in nearly all people, yet it varies individually, both as to extent of distribution and degree of excitability, and even in the same individual at different times. But its extent and limitation offer valuable and unique opportunities for the study of the distribution of the nerve supply, in a manner which no other clinical phenomenon, with which I am acquainted, does so clearly. Thus it reveals certain definite boundaries in the distribution of the spinal nerves considered according to the distribution of each spinal segment. It also shows in suitable cases, the distribution of the nerve supply, considered according to the disposition of the peripheral nerve in a longitudinal or vertical direction, on the dorsal, lateral, and ventral surfaces of the body. It also brings out the dissemination of nervous impulses, in cases of visceral disease. Numerous as my observations have been, they are as yet far from complete; and I am unable to deal exhaustively with the information to be got from the study of this symptom. My object here is to call attention to a valuable symptom, affording a new method of investigation of a subject which has been recently discussed in these pages; I refer to the distribution of cutaneous nerves, and the sensory symptoms associated with visceral disease.

Every one, I suppose, is conscious in their own person of the production of this symptom over large areas of the skin. The sudden exposure of the warm body to the cold air, produces it most extensively. Under certain conditions, apparently consistent with perfect health, the symptom can be produced reflexly by stimulating the skin. The appearances when resulting from such reflex stimulation are either limited to the immediate neighbourhood of the part stimulated, or spread in a diffuse but still definite manner. Thus in stroking the skin firmly with a round, blunt, or broad-pointed instrument of one-quarter to half-an-inch in breadth, the goose-skin appears in the immediate tract of the part stroked and usually extends for some millimetres beyond on either side. (I am speaking now of the skin of the chest and abdomen; other parts do not usually manifest the symptom so readily.) From the area stimulated, the goose-skin may extend over a large tract of skin, but it does so in a very definite manner, the direction varying somewhat in different individuals. Usually it takes place along the areas of distribution of nerves, whose central connections are in close contiguity to those of the nerves supplying the area stimulated, thus manifesting itself on successive fields of skin along a very definite path. When the stimulus is applied to the skin, one can observe each successive tract of skin becoming involved, although it occurs usually with such rapidity that one can scarcely determine the exact limitation at a given moment. In rare cases, the extension is so slow that one can follow it with fair certainty. In the observations I have made on the limited distribution, the methods of stimulation have been usually by stroking the skin with a blunt pencil-case, or by the finger covered with flannel or rough linen. At other times the whole trunk would be gently and equally rubbed with a towel while the goose-skin would appear only in very well-defined areas. I have said that the sudden exposure of the body to the cold atmosphere suffices to produce goose-skin, but it seems as if the slight friction produced in lifting the bed-clothes from the skin, is sufficient to set up a very wide-spread horripilation. I have tried to stimulate with cold, dry and wet—with pieces of ice direct, in an ice bag, and in test tubes—but without result.

So far as my observations go, I have found the goose-skin extend over the whole body, except the ears, hands, and wrists, feet and ankles. In one patient, the raising of the bed-clothes produced it over the whole body with the above exceptions, and also with the exception of the neck and upper part of the chest—an exception of some importance to be dealt with presently.

After the junction of the posterior and anterior roots of a spinal nerve, the nerve divides practically into three divisions which respectively supply the posterior, lateral, and anterior surfaces of the trunk. Among the limb plexuses certain modifications arise, but as my results do not yet permit me to discuss the distribution of the nerves in the limbs from a consideration of the pilomotor nerves, I shall not particularly refer to the extremities in relation to their nerve supply from this point of view. In figs. 5 and 6 of Ross's paper[1] the areas supplied by these divisions of the nerves are mapped out, and agree fairly well with the areas marked out in fig. 1. Voigt[1] had indeed long ago demonstrated somewhat similar areas.

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[1] Ross, J. "On the Segmental Distribution of Sensory Disorders," BRAIN, 1888, p. 344.

The reason I draw attention to this method of studying the distribution of the spinal nerves is because I have found occasionally that stimulation of a limited area of the skin will produce goose-skin, that spreads and fills exactly one or other of these areas. I have under observation two cases in which the goose-skin can occasionally be made to arise only in the area of the distribution of the anterior division, in the one case, and in the area of the distribution of the lateral division, in the other; and in neither case can it be produced in any of the other areas.

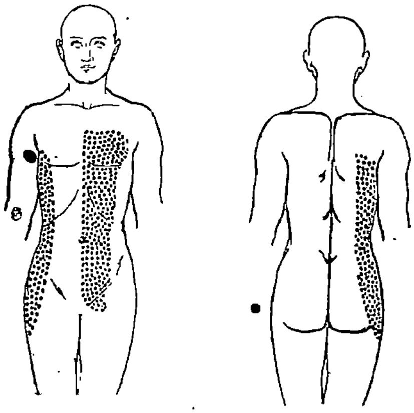

FIG. 1.

On the left side the dotted area represents the distribution of the goose-skin in the region supplied by the anterior divisions of the spinal nerves as in Case 1. On the right side the limits of the distribution of the goose-skin, back and front in the region supplied by the lateral divisions of the spinal nerve, are shown as in Case 2.

Case 1.—Male, aged 30. When the clothes are removed and he lies on his back, the temperature of the room being about 65°, stimulation by stroking the skin transversely in any part of the anterior surface of the left side of the abdomen, produces a typical goose-skin in a quick wave-like manner over the left side of the chest and abdomen. This area is limited *laterally* by a line drawn from the anterior part of the axilla to immediately internal to the anterior superior iliac spine, *internally* by the middle line, *superiorly* by the second rib, and *inferiorly* it fades away into the groin. Stimulation of any other part of the chest or abdomen produces no goose-skin. This area is extremely well-defined, and no difficulty is found in noting its extent. When this patient was stripped for examination in the out-patients' room, the goose-skin was much more universal. But it frequently happens that the areas most responsive to stimulation, vary from time to time.

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[1] Voigt, C. A., "Beiträge zur Dermato-neurologie; nebst der Beschreibung eines Systems neu entdeckter Linien an der Oberfläche des menschlichen Körpers," *Wien*, 1864.

Case 2.—Male, aged 29, complains of pain in his back, the result of a strain five years ago. When any portion of the skin of the chest or abdomen is stroked, the goose-skin spreads wave-like over the following area; it is limited *in front* by a line in the exact situation of that which bounded laterally the goose-skin in the preceding case, and *posteriorly* the limitation is equally abrupt in a line drawn through the angle of the scapula to behind the posterior crest of the ilium. *Superiorly* the goose-skin is lost in the axilla while *inferiorly* it descends over the hip, embracing the skin over the great trochanter (fig. 1).

Nothing could be more striking than the manner in which these areas were marked off. So distinct was the limitation of the goose-skin that one can but attribute it to some central cause. I incline to the view that the stimulation of the skin radiates along the paths that give rise to least resistance; and that in such cases as these, the central connections of the cutaneous nerves appear to be more easily excited longitudinally than transversely. That is to say, the excitation passes with more facility from portions of nerve roots supplying contiguous areas in the anterior or lateral aspect of the skin, as the case may be, rather than spread over the whole field of supply of one nerve. This view is strengthened by the consideration of what happens in the hyper-æsthesia in visceral diseases to which I shall presently refer. More commonly stimulation of a limited area causes the goose-skin to arise and extend by advancing over the whole half of the circumference of the trunk, although even here in its advance it sometimes shows a tendency to affect first, say, the anterior area for a limited extent, next the lateral, then the posterior, and then again a limited extent of anterior area and so on. This, however, occurs so rapidly, that it can occasionally be detected only on its being specially looked for. The usefulness of the goose-skin reflex as a feature for observation is demonstrated in studying the manner in which it is limited, its boundaries presumably coinciding with the areas of distribution of definite nerves. Thus it very frequently happens that, in stimulation of one side of the chest or the abdomen, the goose-skin limits itself to one half of the body, ascending on the chest to a certain extent, then, instead of extending over the clavicles, passing down the arm in a remarkable and instructive manner. But not only does the goose-skin extend in this way, giving information by the limits of its distribution over a large area, but occasionally localized stimulation will produce the phenomenon in distant areas, which we know to be in anatomical relation. Thus stimulation of the upper part of the chest will produce the goose-skin over definite tracts in the arm; again, stimulation of the arm may sometimes produce it in certain definite areas on the chest, front and back. The following case first showed me the importance of the goose-skin as an object for study, and reveals features of such interest, that I quote it in full.

Case 3. — Female, aged 20, admitted into the Victoria Hospital, complains of loss of power in the legs. The patient got wet through three months ago, and her legs felt stiff the following day. Since then her power of walking has gradually become more impaired till now she cannot stand. On careful enquiry, she admits that there was weakness and a feeling of numbness in the left foot and leg, before the date on which she says she got wet. She also admits having had pain in the lower part of the spine. The patient is pale and fairly well nourished. She lies in bed with the legs bent at the knees, the right generally lying upon the left. She cannot voluntarily move the toes and ankle, but can, to a limited extent, move the leg at the knee and hip joints, but this she does stiffly. When forcibly bent, the knee offers considerable resistance, and gives rise to the clasp-knife phenomenon. The knee-jerks are exaggerated on the right side, normal on the left. Ankle clonus is readily developed in both legs. The plantar reflexes are greatly exaggerated, and the lower abdominal reflexes are absent. She has very little power over the bladder and rectum. Over both legs the sensation to touch and pain is impaired and slightly perverted as high as the lower limit of the goose-skin in fig. 2, but there is no distinct border to be made out. The difference between heat and cold cannot be made out in the legs. The muscular sense is impaired. Over the lower third of the sacrum there is a diffuse swelling in the subcutaneous tissues, and the bone here is thickened with an indentation in the spine. The eleventh dorsal vertebra is tender to pressure. Goose-skin appears in certain areas on stimulating the skin. Thus, in testing for the sense of touch on the left side, by lightly stroking with a pin-head from the groin upwards along the abdomen, when the skin midway between the symphysis pubis and umbilicus was reached, the goose-flesh appeared over the abdomen at the part stimulated, and quickly spread in a wave-like manner up the chest, and into the left arm, over an area like that dotted in fig. 2. The lower limit extended irregularly round

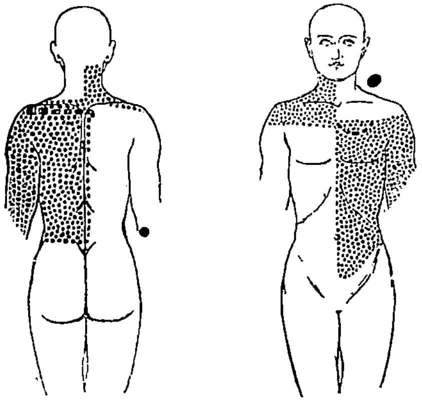

FIG. 2.

The dotted area on the left side represents the distribution of the goose-skin in Case 3. The portions above the interrupted lines in front and behind are those that were produced on stimulating the inner surface of the left upper arm. On the right side the dotted area represents the goose-skin in the distribution of the cervical plexus as in Case 5.

to the second lumbar vertebra. The lower margin was abruptly limited, and before it could disappear it was marked by a dermographic pencil presenting the same peculiar limitation as in fig. 2. The upper limit presented an interesting variation. As one watched the goose-skin ascend, it reached the level of the nipple, coursed round the upper border of the axilla, and descended down the inner surface of the arm for a short extent; then it reappeared on the chest over an area extending from the level of the nipple to the first rib, at the same time descending further down the inside of the arm, and appearing on the back and outer side of the arm, not affecting the forearm nor ascending into the shoulder. If after it had subsided, we repeated the stimulation and observed the progress in the back, we noticed that when the goose-skin reached the level of the second dorsal vertebra, from which the upper limit extended outwards in a straight line, the goose-skin appeared next on the arm, then suddenly reappeared as a band above the previous limit, as high as the seventh cervical vertebra, while it extended lower on the arm and appeared on its outer and posterior aspect.

As it was evident that the goose-skin followed the distribution of the spinal nerves, numerous observations were made of the results of localized stimulation. It was not always easy to make out what the result was, except in one instance, which was frequently verified. If the inner surface of the upper arm was stroked (at various points which it was difficult to localize accurately) there appeared areas of goose-skin on the chest and the back, in the places situated above the interrupted lines drawn in fig. 2, representing presumably the distribution of the second dorsal nerve. Sometimes on the back and front of the chest, on stimulation of the anterior surface of the arm, the goose-skin appeared on the chest in a rounded patch over the second, third, and fourth costal cartilages, next round the nipple, then higher up, from the outer side of the breast to the anterior fold of the axilla; leaving an unaffected area above the nipple of about two inches in breadth. Stimulation of the inner surface of the arm would then produce the goose-skin in this unaffected area, followed by the extension over the whole half of the chest as in fig. 2. Stimulation of the outer aspect of the arm produced no goose-skin on the chest. These phenomena could be elicited also on the right side of the chest, except that the goose-skin never appeared so high on the outer aspect of the arm as it did on the left.

I was inclined at first to consider this excitability of the pilomotor reflex as dependent on the lesion in the cord; but that this is not so is evident from the study of a number of other cases with presumably healthy centres, of which the following is a most noteworthy example:—

Case 4.—Male, aged 35. Admitted to the Victoria Hospital on August 25, 1893, with a compound comminuted fracture of the lower third of the femur. On October 25 it was found that there was no union, and the leg was encased in plaster of Paris. The patient is a man of ruddy countenance, well-nourished, and of powerful muscular development. Except for the fracture, he is in perfect health. In him the goose-skin is extremely active on first exposure of the skin to the air. After it has subsided, stimulation of the skin on the outer surface of the abdomen readily excites it, spreading rapidly over the left side of the chest and down the inner side of the arm and ulnar border of the forearm to the wrist. It leaves unaffected the radial border of the arm, but spreads over the outer and back part of the upper arm. The limits of the eruption are shown in fig. 3. On other occasions I found that stimulation of the skin of the chest as low as the epigastrium on the front and side, produced the goose-skin over a limited area on the inner side of the arm, but not on the chest. This was repeatedly verified. There were some sensory phenomena produced, to which I shall refer later.

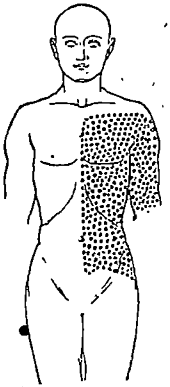

FIG. 3.

Distribution of goose-skin on stimulating any portion of the chest or abdomen on the left side. Case 4.

One very striking feature in these two cases, is the limitation of the area affected by the goose-skin. In the meantime I will only deal with the upper limit on the chest which is practically the same not only in both these cases, but also in a number of others I have observed.

It is evident, I think, that we have here the demarcation of the upper limit of the distribution of the dorsal nerves in the chest, the area above this limit being supplied by the cutaneous branches of the cervical plexus. It happens, not infrequently, that one meets with cases in which the goose-skin is manifested only in the neck and upper part of the chest. The following is a fair specimen of this class:—

Case 5.—Female, aged 31; examined October 13, 1893, was seized yesterday with shivering and sore throat. The tonsils are slightly swollen, and inflamed with pus exuding from numerous follicles. As there was a case of scarlet fever in the house I exposed the chest to see if there was any rash, when I was struck with a well-marked appearance of the goose-skin that arose all over the neck and upper part of the chest. It was limited above to the hair-roots and under the ear and chin, while below it descended in front as low as the level of the second costal cartilage at the sternum, whence it extended outwards in a straight line reaching the level of the third rib in the nipple line, to the shoulder. Behind, the inferior boundary extended along the level of the first dorsal vertebra (see the dotted area on the right side of fig. 2). The goose-skin was much more easily excited on the *right* side than on the left; and the *right* tonsil was the more severely affected. After a few days the throat condition improved, but the goose-skin could still be obtained, though not so readily as before. A fortnight later the throat was still a little painful, and a faint goose-skin could still be perceived in response to stimulation. During the whole time the patient was under observation, it never tended to spread to any other region.

Before entering more minutely into the question how far the goose-skin reflex assists in determining the fields of cutaneous nerve-distribution, there are a few points which require elucidation, before we can confidently employ this phenomenon for such a purpose. At present I will do little more than indicate two of these points, namely, the question of overlapping, and the relation of the goose-skin to areas of skin exhibiting certain sensory disorders.

In regard to the question of overlapping, the study of this subject offers a very conclusive answer, so far as it concerns the pilomotor nerves. When the goose-skin is limited to one side, two conditions are present in regard to its relation to the middle line. If it be well-marked, it distinctly crosses the middle line and affects the opposite side to an appreciable extent. This varies not only in different individuals, but in different places on the same individual. Thus in Case 3, in the epigastrium the goose-skin extended irregularly for one inch to the right of the middle line, when it was well-marked in the left half of the body. Above and below the epigastrium it extended across from one-quarter to half-an-inch. The method employed to demonstrate this with certainty, was to induce the goose skin in the left half of the body, then quickly to mark its boundary by the dermographic pencil. The right side was next stimulated, and the limits of the goose-skin thus produced mapped out in the same manner. It was found that a narrow strip of varying breadth down the centre of the body was affected, when the phenomenon appeared on either side. This overlapping could also be inferred from the further fact that, where the goose-skin on one side was but scantily present, it never quite approached the middle line; so that on stimulating one side, then the other, there was a narrow strip down the middle of the body in which the goose-skin did not appear. This was usually most evident on the back, where the goose-skin does not always manifest itself so distinctly as it does on the anterior aspect of the trunk. On account of this overlapping, the exact fields cannot be marked out on the skin of the body, where neighbouring areas of skin are supplied by neighbouring nerve roots. But when neighbouring areas of skin are supplied by nerve roots whose spinal origins are wide apart, then a very good opportunity is afforded for this method of studying the question of overlap. Thus the skin in the upper part of the chest is supplied by the branches of the cervical plexus, being conterminous with the area supplied by the second and third dorsal nerves—the intervening nerves supplying the arm. I have pointed out the striking manner in which the wave-like production of the goose-skin passes from the chest into the arm, leaving the upper part free. Now it so happens that this limit of distribution is very common and can be readily mapped out. On the other hand, it is not uncommon to find the goose-skin limited to the distribution of the cervical plexus. While, therefore, I have not been able to demonstrate these independent areas in one individual, I have made careful observations of the exact limits in a number of people who presented either the one or the other localization. In these I found a remarkable agreement. In front the upper limit, where the chest was affected, extended usually to the level of the first costal cartilage and from thence outward to the top of the shoulder (figs. 2 and 3). On the other hand, where the area supplied by the cervical plexus was carefully marked out, it descended as low as the level of second costal cartilage and ran out transversely to the shoulder a little below the acromial process (fig. 2). Similarly behind, the upper limit of the goose-skin, when the chest was affected, ascended as high as the seventh cervical vertebra (fig. 2), while the lower limit reached as low as the first dorsal vertebra (fig. 2). These limits were not very abrupt, and at the margins the goose-skin faded away; but I have marked the limits where it could be demonstrated to exist without any doubt.

I have endeavoured to find out if there be any relationship between areas of disordered sensation and those exhibiting the pilomotor reflex. In Case 3, where the area of anæsthesia affected the legs and the lower part of the trunk, there certainly does appear to be a distinct connection, inasmuch as the reflex does not extend much beyond the limits of the healthy skin. But while the area invaded by the goose-skin is very distinctly limited here, that of the anæsthesia is not so distinct, there being a border of slight but varying breadth where it is difficult to determine whether the sensation is normal or otherwise. This border follows in its general contour the line round to the spine, indicating the lower limit of the goose-skin area. The latter never spreads into the parts where the anæsthesia is undoubted and permanent. From my investigations into the relationship of the pilomotor reflex with the hyper-æsthesia and pain of visceral disease, no definite conclusion can so far be drawn; but certain phenomena would lead one to expect that, in some cases at least, very instructive results may yet be obtained. Thus in a case of aortic disease, with well-marked attacks of angina pectoris in the left chest and arm, the hyperæsthesia affects the front of the chest from the level of the second rib to the epigastrium. Behind the anterior axillary line the hyper-æsthesia becomes less distinct, its lower border slanting upwards to the spinal column about the level of the the third dorsal vertebra. The hyper-æsthesia also extends down the inner surface of the upper arm, ulnar border of forearm, to the little finger. The pilomotor reflex, when induced by gently rubbing the abdomen and chest of both sides, appears only on the left side. Its upper limit is situated as high as the second costal cartilage, and extends thence to the inner side of the upper arm, on which it descends as low as the elbow. But the upper limit of the goose-skin on the chest does not correspond exactly to the area mapped out by the hyper-æsthesia, inasmuch as the latter extends outwards to the axilla in an almost straight line from the second costal cartilage, while the upper limit of the goose-skin area curves down to below the third rib in the nipple line, rising again to the same height as the hyper-æsthesia in the anterior axillary fold. Below the goose-skin extends down the abdomen, fading away in the thighs in a manner too indefinite to be mapped out with certainty. No cutaneous hyper-æsthesia exists, and no goose-skin can be induced, in the area supplied by the cervical plexus.

In a case where there were a number of somewhat obscure symptoms which at first I thought were probably symptomatic of erythromelalgia, there were a number of patches of hyper-æsthesia following certain definite nerve tracts in the trunk and limbs, which it is not necessary to my purpose to give here in detail. Stimulation of the skin produced the goose-skin on the chest and abdomen, limited above as in figs. 2 and 3, and this was also the upper limit of an area of hyper-æsthesia that affected the chest. But the point to which I desire to draw attention here, is that when I stroked the skin with a blunt pencil, beginning on the side of the neck, downwards over the middle of the clavicles and down the chest to the level of the nipple, there appeared in the tract of the pencil a streak of goose-skin beginning abruptly at the line previously marked as the upper limit to which the goose-skin and hyper-æsthesia extended, that is, at the level of the second rib.

Above the second rib no signs of the goose-skin could be observed, neither on the chest nor on the neck. This experiment was repeated many times and always with the same result.

That the pilomotor nerves over a limited area may be rendered abnormally sensitive in cases of disease of an internal organ, in the same manner as the sensory nerves, appears likely from Case 5, and such observations as the following:—

Case 6.—Female, aged 59, has suffered from severe pains across the upper part of the abdomen occurring at intervals for the past twelve years. During the last three years she has suffered with increasing frequency, and for the last three months she has scarcely been without pain. She also suffers frequently from attacks of jaundice. I saw her in consultation, and as the symptoms pointed to gall-stone disease, recommended the advisability of an operation, for which purpose she was admitted into the Victoria Hospital, on October 31, 1893. Since her admission she has remained quite free from pain, and the jaundice from which she suffered is gradually disappearing. The attacks of pain are localized across the middle line of the abdomen, midway between the umbilicus and ensiform cartilage, extending well round to the right side. There is no cutaneous hyper-æsthesia; but there is an area of tenderness of indefinite extent, elicited by pinching the deeper structures of the abdominal walls (subcutaneous fat and muscles). On lightly rubbing the chest and abdomen with a piece of flannel, the goose-skin appears in a band three inches broad, extending across the abdomen above the umbilicus, to the left as far as the anterior axillary line, and to the right as far as the middle axillary. The area widens out on the right side, extending over the chest as high as the nipple, but not passing further back. A week after this observation, the patient having still been free from pain and the deep tenderness having nearly disappeared, the area of goose-skin obtained by stimulation was limited entirely to the right side, and covered an area less extensive than in the previous observation.

In another case of gall-stone disease under observation a somewhat similar relationship of exalted sensibility of the external body-wall and of the excitability of the pilomotor reflex can be demonstrated.

In several other cases, I have observed areas of goose-skin limited to regions in which pain and hyper-æsthesia had been felt, but so far I have not yet been able to determine the exact harmony, or otherwise, of these manifestations. There is one feature about the distribution of these areas of goose-skin, that resembles the distribution of the hyper-æsthesia in visceral disease. Frequently the area does not correspond to the distribution of one or more spinal segments in their entirety, but spreads somewhat diffusely into portions of the area supplied by several spinal nerves. In my contribution on the sensory phenomena associated with visceral disease (BRAIN, Autumn Number, 1893, p. 321-374), I pointed out this characteristic both in the hyper-æsthesia of visceral disease and in that associated with herpes zoster. I am not sure whether the irritation tends to spread more along longitudinal paths, affecting the roots supplying the anterior, lateral or posterior aspects of the body (fig. 1), rather than to affect one spinal segment; but I incline to such a view. It may also be that whilst the segmentation of the cord is not so definite as to permit precise division of the cutaneous surface according to nerve supply, excitations spread from any given centre in the cord more readily along paths in a longitudinal than in a transverse direction.

A difference in the distribution of sensory and pilomotor nerves is also evident from the following observation. The chest of a woman whose breast had been excised a year before, presented a long cicatrix about the level of the third rib, extending from the sternum out into the axilla. On exposure of the skin to the air, the goose-skin spread over the chest and arm; but while it extended down to the upper margin of the cicatrix, it left quite free a band of about one-and-a-half inches below and parallel with the cicatrix. On testing the sensation of the skin below I found it quite normal. In another case, an adenoid tumour was removed from the breast. After stitching up the wound (the incision being about five inches in length and situated horizontally near the anterior axillary line), I observed the goose-skin spread over the whole chest, leaving an area about an inch broad internal to the line of incision quite unaffected; the skin was afterwards found quite normal. The sensation caused by, or rather associated with the production of goose-skin, appears to be peculiar in its nature. Most people are acquainted with the sensation; and in Case 4 I made a curious observation. Once while testing for the goose-skin on the chest, I was looking for it near the point of stimulation. The patient called my attention to the fact of its production on the inner side of the upper arm. I asked him how he knew, and he said he felt it coming. On going further into this point, I found that, on certain areas of the chest being stimulated, he felt the sensation extend down the arm to the little finger where no goose-skin was visible. I asked him what kind of sensation it was, and he said, "It is a curious sensation as if goose-lumps were coming out." But when I called his attention to the fact that there was no such appearance on his wrist and hand, he examined the part and appeared surprised. Being somewhat doubtful of his veracity, I put him to the following test. I lightly stimulated the skin by drawing the pencil case transversely across the skin of the neck, gradually descending over the clavicle on to the chest; he said that no sensation whatever was produced; but directly I touched the skin below the first rib, he at once exclaimed that he experienced the sensation. It was also noticed that the sensation arose exactly where the line was crossed, which I had marked along the upper limit of the goose-skin on the chest. Similiarly, on testing the skin from the level of the umbilicus and gradually ascending whenever I touched the skin above the nipple, the same sensation was experienced. Excitation of any area between the nipple and first rib produced the feeling. The latter always quickly spread onwards from the chest to the inner surface of the arm, and down the ulnar border of the forearm to the little finger, occasionally with the accompaniment of goose-skin on the inner surface of the arm. The patient could have no knowledge of the distribution of the spinal nerves; but his sensation indicated their course so distinctly that I have no hesitation in giving credence to his expressions. The experiments were often repeated on both sides of the body, and these results verified.

My object in writing this paper is merely to call attention to a method of studying the nerve distribution, which is free from many objections that vitiate other methods. I would have liked to have compared the results obtained by clinical investigations, with those of Langley and Sherrington; but my observations are still far too few. From what I have seen, I feel convinced that very instructive results are obtainable, particularly with reference to the nerve distribution in the limbs, and to the diffusion of irritation to nerve centres in visceral disease. I must, however, remain content with presenting these suggestions, other matters preventing me from pursuing this inquiry for the present.

ONE HUNDRED CONSECUTIVE CASES OF EPILEPSY; THEIR REFRACTION AND THEIR TREATMENT BY GLASSES.[1]

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FOR some years past there has been a question constantly put forward as to a relationship between epilepsy and errors of refraction, which up to the present time has not been satisfactorily met.

With a view of collecting some certain information on the subject, I, last year, began examining the eyes of all the cases of epilepsy that I could obtain at the West End Hospital for Nervous Diseases and Paralysis, and treating them as far as possible with correcting lenses. I have the honour of submitting to you the results of this work in one hundred consecutive cases of epilepsy.

In order to remove all possible doubt as to diagnosis, each case has first been seen by the physician in attendance, and subsequently, at my request, passed on to me as being one of true epilepsy.

With regard to the examination of the eyes, I may state that in seventy-four cases sulphate of atropine drops (grs. iv. and grs. ii. ss. ad. ℥i.) were employed; in twenty-three cases hydrobromate of homatropine drops (grs. vi. ad. ℥j.) were used, and in three cases only the process was carried through without a mydriatic. Of these three cases, one was a woman of sixty-seven years of age, with Hm. 1 D., and taking + 4.50 D. for near work; the second, a man with myopia, R.E.—8 D., L.E.—2 D., and the third was a patient, who strenuously objected to the drops, showing Hm. .50 D.

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[1] Read before the Ophthalmological Society and published in this Journal by permission of the Council.

In working out the refractions, exceeding care has been taken to ensure accuracy in the results.

The first table contains the classification of the refractions of the one hundred cases. In this—

(*a*) The total refraction under the mydriatic has been taken.

(*b*) Hypermetropia of .75 D. has been reckoned as emmetropia.

(*c*) Astigmatism of .25 D. has not been included.

In the one hundred cases I have found the results shown in Table A.

Here the simple hypermetropia and the total astigmatism are equal in amount, each having forty-two cases.

Wishing to compare these results with the refractions obtaining in apparently normal eyes, I could find nothing more suitable for that purpose than my own paper, published in the last volume of the *Transactions of the Ophthalmological Society of Great Britain*. From the cases contained in this I have worked out the following percentages on the same lines, as I have done in the epileptic cases (see Table B).

The comparison between the two classes is shown there.

The marked differences exist in the amounts of the simple hypermetropia, and of the astigmatism. Of simple hypermetropia there are twenty-eight cases per cent. *less* in the epileptic than in the apparently normal class.

Of astigmatism of all kinds, there are twenty-six cases per cent. *more* in the epileptic division than in the normal one, this difference being in great part made up by the increase in epileptics of compound hypermetropic astigmatism, viz., eighteen cases more than the normal percentage. If the difference of twenty-six cases per cent. of total astigmatism in the epileptic be added to the forty-two cases per cent. of the simple hypermetropia of the normal class, the result comes to within two cases per cent. of the normal amount of simple hypermetropia, viz., 68. It would almost appear as if the increase of astigmatism in epileptics were taken from the normal amount of hypermetropia.

I beg now to refer to the large table (C); on it will be found each case arranged transversely under its various headings, viz.:

(1) No. of case; (2) age; (3) sex; (4) family history as to fits; (5) age when fits began; (6) frequency &c., of fits; (7) duration of fits; (8) time when fits occur; (9) aura; (10) cephalalgia; (11) vertigo; (12) pupils; (13) fundus examination, &c.; (14) vision and refraction before mydriasis; (15) mydriatic used; (16) vision and refraction after mydriasis; (17) glasses ordered, &c.; (18) length of time glasses worn; (19) condition, &c., while wearing glasses.

Of one hundred consecutive cases of epileptics examined, twenty-five did not need glasses; seventy-five were ordered to wear them, and of these there were twenty-three who either did not wear them or failed to report themselves later, and could not be found. Of the remaining fifty-two patients there are—

(*a*) Thirteen who have had no fits since wearing the glasses, during periods varying from one year down to four months. These are Nos. 5, 11, 24, 25, 33, 52, 57, 58, 66, 78, 87, 90, 91.

(*b*) Three who have remained *in statu quo*, Nos. 3, 46, 72.

(*c*) Thirty-six whose condition has improved since using the glasses—in the majority of these the improvement is in a marked degree. The refractions of the thirteen cases who have had no fits, are shown in the large tables. With the exception of the one case of mixed astigmatism the errors are not great; the proportion of simple hypermetropia to the total astigmatism still stands about as in the larger scale, the compound hypermetropic astigmatism representing the greatest part as before.

It is not necessary for me to weary you with a long account of these cases. I will however read a few of them as examples; others may easily be found in the tables.

Brief histories of some patients who have been free from fits since wearing glasses.

Case I., *No.* 5, *Sept.*, 1892.—A man aged 24 years, a porter by occupation. There is no family history as to fits. His fits began at the age of 21 years, and they have continued up to this month. He has about seven or eight in a month, and very rarely goes a month without an attack. He has often had several fits in rapid sequence. They always occur during the day—never at night. He has no aura. He complains of bad frontal headache and also of occipital pains; the latter are rare. Vertigo is not marked, but he has it occasionally. He has been taking bromide of potassium for a long while.

Oct., 1893.—He has had no fits since wearing his glasses, which were at first ordered for near work only. In July, his headaches still continuing, he was told to carry his spectacles always, and he is now absolutely free from cephalic pain. His general health is much better.

Case II., *No.* 11, *Sept.*, 1892.—A woman aged 20 years, a housemaid. Her mother has had fits since she was 9 years old up to the present date, Sept., 1892. A brother, aged 22 years, has had fits since 6 months. Her fits began at 19 years of age. She has now about one a month, but has had them more frequently. They generally occur at night. She has no aura, though on one occasion she saw "mixed colours" just before a fit. She suffers from vertical headache "dreadfully" and frequently; she also has attacks of vertigo nearly every afternoon. She has been taking bromide of potassium for a long time.

July, 1893.—She has sensations but no fits.

Sept., 1893.—She has slight sensations but no fits.

Case III., *No.* 24, *Nov.*, 1892.—A woman aged 30 years, a servant. There is no family history as to fits. Her fits began at 20 years of age. She has them at variable periods and very often. They occur usually in the morning, but also occasionally at night. She has no aura. She suffers frequently from headache, which is frontal and vertical in situation, also from numerous attacks of vertigo. She has been taking bromide of potassium for a long time.

June, 1893.—She has had no fits and has lost vertigo. The headache is rare and lessened in severity.

Sept., 1893.—No fits—no headaches to speak of—rarely some vertigo.

Case IV., *No.* 25, *Nov.*, 1892.—A youth aged 16 years, a tailor. There is no family history of fits. The fits began in childhood. He now has them about three times a week, usually at night. He has no aura. He suffers from frequent headaches which he calls "awful"; they begin with severe frontal pains, and then extends all over his head. Vertigo is common and usually occurs in the daytime. He has been taking bromide of potassium for a long time.

May, 1893.—No fits since glasses.

Sept., 1893.—No fits, no headaches and no vertigo.

Oct., 1893.—The same.

Brief histories of some cases of marked improvement.

Case I., *No.* 2, *Sept.*, 1892.—A woman aged 46 years, a dressmaker. Her eldest brother had fits between the ages of 14 and 21 years. Her fits began at about 18 years of age. She has one fit at variable periods; now they are much more frequent. She has occasionally gone a few months without an attack. She has them always at night. Her warnings are a "feeling of unsteadiness" in frontal and occipital regions of head. Her headaches are very severe and frequent, chiefly frontal, but also occasionally occipital, and her vertigo is so intense that she reels in her walk sometimes. She has taken bromide of potassium for a long time.

March, 1893.—Has had two fits since glasses; ordered an additional pair for presbyopia.

June, 1893.—No fits, no cephalalgia of importance.

July, 1893.—No fits, slight cephalalgia.

Sept., 1893.—One very slight fit in August. Is much better in general health.

Case II., *No.* 28, *Nov.*, 1892.—A girl aged 15 years. There is no family history as to fits. The fits began at 14 years of age. She has now about one attack a week—often two in rapid sequence. They occur at night towards the early morning. She has no aura. Vertical headache is very frequent and she occasionally has occipital pains. She is never giddy. She has been taking bromide of potassium for a long time. She went from Nov., 1892, quite without fits to Sept. 13, 1893, when she had one attack, being at this time extremely worried and nervous about obtaining a situation.

Case III., *No.* 16, *October*, 1892.—A girl aged 14 years. Her father is believed to have had fits in early youth. Her fits began at 9 years of age, when she had one fit. She was then free from attacks until the beginning of her fourteenth year, when they appeared again. She is now having six or seven fits during the day, and they occur about every hour during the night. She has no aura, and she suffers from very severe and frequent headaches, which are frontal and vertical in position; she has no marked vertigo; she has been taking bromide of potassium for some time. Her mother objected to her reading because she thought the fits were brought on through it.

August, 1893.—No fits since glasses; no cephalalgia; is much better.

September 4, 1893.—Has had two fits during the night; she is near her menstrual period.

September 15.—She has had a few fits at night.

Case IV., *No.* 20, *Oct.*, 1892.—A man aged 20 years, a carpenter. His mother has about one fit a month. His fits began at 16 years of age. He has now about one a week; they occur in the daytime, never at night. He has no aura; he is free from cephalalgia, except just after a fit when it is frontal, but this is nothing marked. There is never any vertigo. He has been taking bromide of potassium for a long time.

May, 1893.—No fits since glasses.

Sept., 1893.—One very slight fit.

In all the cases the physician has, I believe, continued his usual treatment for some time after the glasses had been ordered.

Case II. of this second series is a type of several that I have met with. The proper correction of the error of refraction by glasses evidently arrests their fits, and they do well until some other irritation comes to play. In this case the patient has no fits from the time she begins to carry glasses, from November, 1892, to September, 1893, she then has one fit as a result of nervous excitement produced by a difficulty in obtaining a certain situation. Another patient also has practically the same history, and brings on his fit by foolishly diving from a height into cold water. Another one is free for months and then is nearly run over by a hansom cab, when she immediately has six fits. Another goes many months and has a return of her fits just at her menstrual period. In a great number some such occasion appears to cause a return of the fits, but in no such case have the fits resumed their previous severe character or frequency.

It seems to result that, given a certain condition of instability of the nervous system—

(*a*) Errors of refraction may excite epilepsy.

(*b*) The correction of the errors of refraction will—in combination with other treatment—in many cases cure or relieve the epileptic condition. In this hundred cases forty-nine patients have been cured or relieved.

(*c*) In some cases, when the refraction error has been corrected the epilepsy will continue, generally in a modified form, in consequence of other irritation, even though the error of refraction may have been the exciting cause of the fits in the first instance, as shown in the cases that I have pointed out.

I am strongly of the opinion that in every case of epilepsy—in addition to general treatment and the investigation of other organs—the eyes should be carefully examined under a mydriatic, with a view to correcting any error of refraction that may exist by proper spectacles.

A few other points occur which I wish to notice, viz.:—

1. That in twenty-three cases only could I obtain any family history as to fits.

2. The marked severity and frequency of frontal headache, I found about sixty-five per cent. suffered from this; vertical headache came next in number but never appeared so severe as the frontal form.

3. That in about sixty-seven cases the pupils were more or less dilated, and in a large proportion of these, their reaction was lazy; in about six cases the pupils were more or less contracted, and the remaining twenty-seven patients appeared to be normal in every way.

4. That in five cases I found lens opacities of apparently congenital origin.

5. That pulsating veins and physiological cups were very frequently observed.

TABLE A. **TABLE B.**

Epileptics, per cent. from 100 cases.			TABLE OF COMPARISON BETWEEN EPILEPTICS AND APPARENTLY NORMAL CASES.			Normals, per cent. from 50 cases.
7			Emmetropia			6
42			Simple Hypermetropia			70
6			Simple Myopia			2
42	Total Astigmatism.	3	Simple Hyper., Astigmatism	4	Total Astigmatism.	16
		24	Compound do. do.	6		
		2	Compound Myopic do.	0		
		6	Mixed Astigmatism	0		
		7	Other Astigmatism { marked Anisometropia	6		
3			Other Cases { marked Anisometropia			6
100			**TOTAL**			100

TABLE (C) OF NOTES OF ONE HUN

No of Case.	Sex.	Age.	Family History as to Fits.	Age when fits began	Frequency, &c., of Fits before Examination.	Duration of Fits.	Time when Fits occur.	Aura.	Cephalalgia.	Vertigo.	Pupils.	Fu
1	M	8	Aunt died at 35 years of age from epileptic fits	7	Has now about 5 or 6 fits a week. Has had 5 fits in one day	4 or 5 min.	Matutinal, between 6 & 8 o'clock	Occasionally feels giddy before a fit	Frontal, frequent and severe	Occasionally before a fit	Moderately dilated	Pu vein
2	F	46	Eldest brother had fits from 14 to 21 years of age	18	Has fits at variable periods. Occasionally has gone a few months. Now they are more frequent	A few min.	Nocturnal, never in daytime	Feelings of unsteadiness in frontal region	Frontal and occipital, very severe & frequent	Frequent: reels often in her walk	Moderately dilated	Disc semi logic pul vei pher in b (con
3	M	25	One sister had fits in youth, beginning at 12 years of age	10	Has about 50 fits per annum. Occasionally 3 or 4 in rapid sequence	About 3 min.	Nocturnal	Fit begins by head turning in small jerks to left shoulder	Frontal, not severe and occasional	None	Normal	Pul vei pher bot (con
4	M	16	....	12	....	......	....	....	....	....	....	....
5	M	24	None	21	Has 7 or 8 fits in a month. Rarely goes a month without one. Occasionally 3 or 4 in rapid sequence	2 or 3 min.	Daytime, never nocturnal	None	Frontal, severe and frequent	Occasionally	Dilated, slow reaction	Pul veins phy cupp —nil
6	M	15	None	7	Has 2 fits in a week	5 min.	Matutinal, never nocturnal	Peculiar feeling in eyes	Frontal, not bad; occasional	Rarely	Moderately dilated	N
7	F	45	....	43	....	......	....	....	....	....	....	.....
8	F	17	None	11	Has about 4 fits in a month. Occasionally 2 or 3 in rapid sequence	2 or 3 min.	Nocturnal	Pains in legs, and occasionally sees patches of green going into red	Frontal, frequent and severe, especially bad before a fit	Frequent	Dilated	Discs semic logica Y-s opaci ant., lense
9	F	7	None	3	Has 3 or 4 fits daily for some days, then perhaps goes a month. Cannot walk. Sits where placed and never moves	A few min.	Nocturnal & day	None	Frontal, frequent and severe		Moderately dilated	N
10	F	35	None	17	Daily	A few min.	Nocturnal, rarely in daytime	Sensations in right arm	Frontal and occipital rare, & only after a fit not severe	Very rarely	Normal	N
11	F	20	Mother has had fits since 9th year, one brother since 22nd year (6 mos.)	19	Has fits about once or twice a month, has had them more frequently	10 min.	Nocturnal	None (once had "mixed colors" before eyes)	Vertical, frequent and 'dreadfully'	Often, nearly every afternoon	Moderately contracted	Discs mic, pul veins
12	F	17	None	15	Has about once in 3 months an attack of 20 or 30 fits in one week	10 min.	Nocturnal & daytime	None	Frontal	Matutinal on rising	Moderately dilated	Pulsat physi

ECUTIVE CASES OF EPILEPSY.

893.

Refraction ydriatic.	Mydriatic used.	Vision and Refraction after Mydriatic.	Description of Glasses Ordered.	Length of time Glasses were in use.	Condition since wearing Glasses.
n.i. let n.i.	Gutt. Atrop. Sulph. grs. iiss. ad ℥ i. thrice daily for 3 days	$<\frac{6}{60}+3.50\text{ D.}=\frac{6}{6}$ $<\frac{6}{60}+4.50\text{ D.}=\frac{6}{6}$	$\left.\begin{matrix}+2.50\text{ D.}\\+3.50\text{ D.}\end{matrix}\right\}$ for constant wear	3 mos.	JULY—Fits are many less in number. Cephal. and vertigo better. SEPT.—Still better. Mother has not kept an account, but he often goes many days without a fit (glasses not obtained until months after being ordered)
2.75D. = $\frac{6}{12}$	Do. grs. iv. ad ℥ i	$\frac{6}{18}+\begin{matrix}+1.50\text{ D.}\\\text{emm.}\end{matrix}=\frac{-.75\text{ D.}=\frac{6}{6}}{+1\text{ D. cyl. ax.}-}$ $\frac{6}{18}+\begin{matrix}+1.75\text{ D.}\\\text{emm.}\end{matrix}=\frac{0\quad=\frac{6}{6}}{+1\text{ D. cyl. ax.}\diagdown 120^\circ}$	(1) Less +1 D. sph. for distance. (2) c̄ +2 D. sph. for near work	12 mos.	During the first 6 months she had glasses for distance only, and had 2 fits then. Subsequently she had another pair of spectacles for near work for presbyopia, since when she has had one slight sensation rather than a fit, and her bad headaches are almost gone. Vertigo is also rare. Is much better and stouter
$\frac{6}{18}$ n.i. $\frac{6}{24}$ n.i.	Do. 7 days	$<\frac{6}{60}+\begin{matrix}+1\text{ D.}\\+2.50\text{ D.}\end{matrix}=\frac{+1.75\text{ D.}=\frac{6}{6}}{+1.75\text{ D. cyl. ax.}\mid\text{v.}}$ $<\frac{6}{60}+\begin{matrix}+.75\text{ D.}\\+3\text{ D.}\end{matrix}=\frac{+1.50\text{ D.}=\frac{6}{6}}{+2.50\text{ D. cyl. ax.}\mid\text{v.}}$	Ordered less + 1 D. sph. for constant wear	8 mos.	In MARCH fits not so severe in character. During 5 months before glasses 21 fits, during 5 months after 12 fits. In MAY case died—cause unknown
let n.i. do.	Do. 3 days	$\frac{6}{12}\text{ let}+\begin{matrix}+1.75\text{ D.}\\+2\text{ D.}\end{matrix}$ $\frac{6}{18}+\begin{matrix}+2\text{ D.}\\+1.75\text{ D.}\end{matrix}$	No glasses		
m. 1 D. = $\frac{6}{6}$ m. 1 D. = $\frac{6}{6}$	Do	$+2.75\text{ D.}=\frac{6}{6}$ $+2.75\text{ D.}=\frac{6}{6}$	+ 1.75 D. sph. for near work, then constant	12 mos.	Has had no fits since wearing glasses. Glasses were first ordered for near work only, but because of cephal. he was told to carry them constantly, since when cephal. has entirely disappeared
m. 1 D. = $\frac{6}{6}$ do.	Do.	$\frac{6}{12}+1.75\text{ D.}=\frac{6}{6}$ $\frac{6}{24}+1.75\text{ D.}=\frac{6}{6}$	+1 D. constant wear	11 mos.	JUNE—Glasses confirmed. JULY—One slight fit. Has worn glasses for near work only
n. .50 D. = $\frac{6}{6}$ m. 1 D. = $\frac{6}{6}$	Homatropine	$+1\text{ D.}=\frac{6}{6}$ let $+1.75\text{ D.}=\frac{6}{6}$	$\left.\begin{matrix}+2\text{ D.}\\+2.50\text{ D.}\end{matrix}\right\}$ For near work		
$\frac{6}{6}$ n.i. $\frac{6}{12}$ n.i.	G. A. S. grs. iv. 3 days	$+\begin{matrix}+2\text{ D.}\\+1.25\text{ D.}\end{matrix}=\frac{+1.25\text{ D.}=\frac{6}{6}\text{ ii.}}{+.50\text{ D. cyl. ax.}\diagup 110^\circ}$ $+\begin{matrix}+1.25\text{ D.}\\+2\text{ D.}\end{matrix}=\frac{+1.25\text{ D.}=\frac{6}{6}\text{ iii.}}{+1\text{ D. cyl. ax.}\diagdown 150^\circ}$	Cylinders only for constant wear	6 mos.	JUNE—Has had 4 fits only since wearing glasses, these all upon one day. SEPT.—Had 1 fit JULY 1st and 1 AUG. 19th. Does not wear spectacles constantly
not read.	Do. grs. iiss. ad ℥ i. 1 week	$+\begin{matrix}+4\text{ D.}\\+2.50\text{ D.}\end{matrix}=\frac{+2.25\text{ D.}}{+1.50\text{ D. cyl. ax.}\mid\text{v.}}$ $+\begin{matrix}+4\text{ D.}\\+2.25\text{ D.}\end{matrix}=\frac{+2\text{ D.}}{+1.50\text{ D. cyl. ax.}\mid\text{v.}}$	Ordered less + 1 D. sph. for constant wear	12 mos.	JUNE—Decidedly better, walks fairly well, is fatter and better tempered. Fits less in number and all of lessened severity. SEPT.—Has been 4 months without a fit. Has recently had 3 fits. Is now more intelligent and altogether better in health
$\frac{6}{6}$ n.i. $\frac{6}{6}$ iv. n.i.	Homatropine	$+1\text{ D.}=\frac{6}{6}$ $+1\text{ D.}=\frac{6}{6}\text{ iii.}$	No glasses		
1. 1 D. = $\frac{6}{6}$ let. 1. 1 D. = $\frac{6}{6}$ let.	G. A. S. grs. iv. ad ℥ i. t. d. ut. 3 days	$\frac{6}{36}=+\begin{matrix}+1.50\text{ D.}+1.50\text{ D.}\\+2.50\text{ D.}+1\text{ D. cyl. ax.}\end{matrix}\begin{matrix}\text{R.}\diagdown 45^\circ\\=\frac{6}{6}\text{ iv.}\\\text{L.}\diagup 55^\circ\end{matrix}$ $\frac{6}{36}=$ Do.	Ordered less + 1 D. sph. for constant wear	12 mos.	JULY—Still has sensations, but no fits. SEPTEMBER—no fits, still sensations
$\frac{6}{6}$ n.i. $\frac{6}{6}$ n.i.	Do.	$+\begin{matrix}+.75\text{ D.}\\+1\text{ D.}\end{matrix}$ Do.	No glasses		

No. of Case.	Sex.	Age.	Family History as to Fits.	Age when fits began	Frequency, &c., of Fits before Examination.	Duration of Fits.	Time when Fits occur.	Aura.	Cephalalgia.	Vertigo.	Pupils.	Fu
13	F	16	None	6	Has one or more fits daily	15 min.	Nocturnal, rarely in daytime	None	Frontal, very severe and frequent	None	Normal	
14	M	27	....	19	One fit in 3 days, has gone 14 days without one	—	Nocturnal	None	—			
15	F	16	....	15	....	......	....	....	....	....	....	....
16	F	14	? Father when a child	9	One fit at 9 years of age, no more till 14. Began again and still continue. Hourly during night and 6 or 7 in the daytime	A few min.	Nocturnal & in daytime	None	Frontal and vertical, very severe and frequent, also very bad after a fit	None	Normal	Puls
17	F	39	None	25	Has about 1 fit every 3 weeks, formerly more frequent	A few min.	Formerly nocturnal, now nearly always in the daytime	None	Not often frontal, shooting neuralgic pains	None	Moderately dilated	Puls R. whi cup —dc mic
18	F	18	Mother had fits when a child	13	Has 3 or 4 fits a week	5 min.	Nocturnal, rarely in daytime	None	None, a "dulness" after a fit	None	Dilated, slow reaction	Puls ma regu edge
19	F	36	None	16	Has about 15 fits a year. Formerly has had 8 fits a day	10 min.	Nocturnal, rarely in daytime	Frontal cephalalgia, has seen lights before a fit	Frontal very bad before a fit	Formerly just before fits, now less	Moderately dilated	Disc mic phy ping ting
20	M	20	Mother has about 1 fit a month	16	Has about 1 fit a week	10 min.	Matutinal	None	None, except after a fit, then frontal but not bad	None	Dilated	Phys ping ting
21	M	41	One brother went insane and died from fits	32	Has had about 16 fits in all	A few min.	Matutinal	None	Vertical, but not marked	Very rarely	Normal	Pulsa
22	M	26	None	13	Has 5 or 6 slight fits daily & several bad ones during a week	2 or 3 min.	Matutinal, also nocturnal	None	Frontal, severe, also occipital	Constantly, & then his sight "appears to go"	Moderately dilated	
23	F	35	None	30	Has 2 or 3 fits a day; has had 7 in one day. Rarely goes a month without an attack	Under ½ hour	Matutinal	Feeling in right hand and leg occasionally	Frontal, always bad	Constantly, nearly every morning	Moderately dilated, slow reaction	Discs pul veins
24	F	30	None	20	Has fits at variable periods, but frequently	A few min.	Matutinal, also nocturnal	None	Frontal and vertical frequent	Frequent	Moderately dilated	P l veins
25	M	16	None	In babyhood	Has fits about 3 times a week	A few min.	Nocturnal	None	Frontal, extending all over; at all times "awful"	Very frequent	Dilated, slow reaction	Pul veins
26	M	21	None	15	Has from 1 fit a week to 1 in two months	A few min.	Matutinal	None	None	None, unless when excited	Moderately contracted	Pul veins Cent cup.
27	F	44	None	29	Has 3 or 4 fits a day, with occasional intervals of a week or sc	3 or 4 min.	Daytime, occasionally nocturnal	Sensations in legs occasionally	Pressure on vertex	Frequent & severe	Normal	

Refraction ydriatic.	Mydriatic used.	Vision and Refraction after Mydriatic.	Description of Glasses Ordered.	Length of time Glasses were in use.	Condition since wearing Glasses.
n.i. n.i.	Do. grs. iiss. ad ℥ i. do.	$\frac{6}{24}+\begin{matrix}-.75\text{ D.}\\+3.50\text{ D.}\end{matrix}=\frac{+3.50\text{ D.}=\frac{6}{12}}{-4\text{ D. cyl. ax.}-}$ $\frac{6}{36}+\begin{matrix}+4\text{ D.}\\-.75\text{ D.}\end{matrix}=\frac{+4\text{ D.}=\frac{6}{12}}{-4\text{ D. cyl. ax.}-}$	Ordered less + 1 D. sph. for constant wear	10 mos.	JULY—No fits until DECEMBER, when she had rheumatic fever, therefore discontinued spectacles for more than 14 days. The fits returned almost daily for some weeks and then slowly disappeared. She has now been some weeks free. Cephal. is rare now
t n.i. t n.i.	Homatropine	+ 1.25 D. = $\frac{6}{6}$ let Do.	No glasses		
n.i. n.i.	G. A S. grs. iv. ad ℥ i. t. d. ut. 3 days	$\frac{+2.75\text{ D.}=\frac{6}{6}\text{ let}}{+4\text{ D. cyl. ax.}-}$ $\frac{+2.75\text{ D.}=\frac{6}{6}}{+.50\text{ D. cyl. ax.}-}$	Ordered less + 1 D. sph. for constant wear		
n.i. n.i.	Do. 7 days	$\frac{6}{36}+\begin{matrix}-?\\+4.50\text{ D.}\end{matrix}=\frac{+4.50\text{ D.}=\frac{6}{12}}{-5\text{ D. cyl. ax.}-}$ $\frac{6}{60}+\begin{matrix}-5\text{ D.}\\-1\text{ D.}\end{matrix}=\frac{-.50\text{ D.}=\frac{6}{12}}{-4\text{ D. cyl. ax.}-}$	$\frac{+3.50\text{ sph.}}{-5\text{ D. cyl. ax.}-}$ $\frac{-1.50\text{ D.}}{-4\text{ D. cyl. ax.}-}$ For constant wear	12 mos.	MARCH—No fits since glasses no cephal., feels well, looks bright and lively. OCTOBER—Had 1 fit on 4th September, 1893, had 2 during the night, having previously had no fits since glasses, that is, during 11 months. She began fi s again at a menstrual period, and they still occur occasionally but are slight in character
n.i. n.i.	Homatropine	$\frac{6}{36}\times\begin{matrix}+3.50\text{ D.}\\\text{emm.}\end{matrix}=\frac{0}{+3\ 50\text{ D. ax.}\diagdown 45^\circ}=\frac{6}{12}$ $\frac{6}{36}\times\begin{matrix}+3.50\text{ D.}\\-1.50\text{ D.}\end{matrix}=\frac{0}{+3.50.\text{ D. ax.}\diagup}$ $\frac{6}{18}$ let	No glasses (afraid to wear them)		
.50 D. = $\frac{6}{6}$ Do.	G. A. S. grs. iv. ad ℥ i. t. d. ut. 3 days	$+\begin{matrix}+1.50\text{ D.}\\+.75\text{ D.}\end{matrix}$ $+\begin{matrix}+1.25\text{ D.}\\+1.75\text{ D.}\end{matrix}$	No glasses		
.50 D. = $\frac{6}{6}$ Do.	Homatropine	+ 1.25 D. = $\frac{6}{6}$ + 1.25 D. = $\frac{6}{6}$ iv.	No glasses		
n.i. n.i.	G. A. S. grs. iv. ad. ℥i. t.d. ut. 3 days	$+\begin{matrix}+.75\text{ D.}\\+2\text{ D.}\end{matrix}=\frac{+.75\text{ D.}=\frac{6}{6}}{+1.25\text{ D. ax.}\vert\text{ v.}}$ $+\begin{matrix}+.75\text{ D.}\\+2.25\text{ D.}\end{matrix}=\frac{+.75\text{ D.}=\frac{6}{6}}{+1.25\text{ D. ax.}\diagup 100^\circ}$	Ordered cylinders only for constant wear	12 mos.	Had no fits from time of wearing glasses (for 11 months), when he had 1 slight fit. Feels much better
1 D. = $\frac{6}{6}$ let Do.	Homatropine	$\frac{6}{12}+\begin{matrix}+1.25\text{ D.}\\+1.50\text{ D.}\end{matrix}$ $\frac{6}{6}$ let Do.	No glasses		
1 D. = $\frac{6}{6}$ Do.	G. A. S. grs. iv. ad ℥ i. t. d. ut. 3 days	$+\begin{matrix}+2.75\text{ D.}\\+3.75\text{ D.}\end{matrix}=\frac{+2.75\text{ D.}=\frac{6}{6}\text{ let}}{+1\text{ D. cyl. ax.}\vert\text{ v.}}$ $+\begin{matrix}+2.75\text{ D.}\\+3.50\text{ D.}\end{matrix}=\frac{+2.75\text{ D.}=\frac{6}{6}}{+.75\text{ D. cyl. ax.}\vert\text{ v.}}$	Ordered less + 1 D. sph. for constant wear		JUNE—Has had no fits since atropine drops were begun. Cephal. better
$\frac{6}{60}$ n.i. $\frac{6}{60}$ n.i.	Do.	$<\frac{6}{60}+\frac{+1.75\text{ D.}=\frac{6}{12}}{-4\text{ D. cyl. ax.}\diagdown 50^\circ}$ $<\frac{6}{60}\ \frac{+1\text{ D.}=\frac{6}{12}\text{ let}}{-3\text{ D. cyl. ax.}\diagup 60^\circ}$	Ordered cylinders only for constant wear		Glasses obtained 14th April, 1893. JULY—Has had 4 fits since glasses were worn, and cephal. is very much better. Vertigo is rare. SEPTEMBER—Three fits; very much better in health
ii. n.i. iii. n.i.	Do.	$<\frac{6}{60}+\begin{matrix}+2.25\text{ D.}\\+4.50\text{ D.}\end{matrix}=\frac{+2.25\text{ D.}=\frac{6}{6}}{+2.25\text{ D. cyl. ax.}\vert\text{ v.}}$ $<\frac{6}{60}+\begin{matrix}+2.50\text{ D.}\\+4.50\text{ D.}\end{matrix}=\frac{+2.25\text{ D.}=\frac{6}{6}}{+2\text{ D. cyl. ax.}\vert\text{ v.}}$	Ordered less + 1 D. sph. for constant wear	11 mos.	Is better. Has had no fits since wearing glasses. Cephalalgia is very rare. Vertigo has almost gone
1 D. = $\frac{6}{6}$ let Do.	Do.	$\frac{6}{24}$ + 3 D. = $\frac{6}{6}$ $\frac{6}{36}$ + 3 D. = $\frac{6}{5}$	+ 2 D. for constant wear	11 mos.	Is better. No fits since glasses, no cephalalgia and no vertigo
1.50 D. = $\frac{6}{6}$ Do.	Do.	$\frac{6}{24}$ + 3.50 D. = $\frac{6}{6}$ let $\frac{6}{18}$ let Do. = $\frac{6}{6}$ let	+ 2 D. for constant wear		SEPTEMBER—Is much better, but has not worn glasses constantly until after JULY, since when he has had no fits
.50 D. = $\frac{6}{6}$ iii. .50 D. = $\frac{6}{6}$ ii.	None	—	+ 1 D. for near work		

No. of Case.	Sex.	Age.	Family History as to Fits.	Age when fits began	Frequency, &c., of Fits before Examination.	Duration of Fits.	Time when Fits occur.	Aura.	Cephalalgia.	Vertigo.	Pupils.	Fu
28	F	15	None	14	Has 1 fit a week, often 2 or 3 in rapid sequence	10 min.	Nocturnal & early morning	None	Vertical frequent, occipital occasional	None	Dilated	P u vei
29	F	34	None	22	Has about 1 fit in 14 days	A few min.	Nocturnal, also in daytime	Heavy and sleepy before fits	Frontal very frequent, also universal after a fit	Frequent	Normal	P u vei Ph
30	F	17	None	13	Has about 1 fit a month, at variable periods	15 min.	Both in daytime and night-time	None	Frontal, bad after a fit	Not marked]	Dilated, slow reaction	P u vei ph
31	F	16	Maternal grandmother had fits which stopped at 16 years of age	13	Has from 2 fits a day to 2 a week. Has gone 2 mos. without a fit	10 min.	Nocturnal, also in daytime	None	Frontal frequent and severe, occasional temporal	None	Moderately dilated	
32	M	16	None	12	Has 1 fit daily, occasionally has attacks of many fits and then goes a week or so without any	5 min.	Nocturnal, also in daytime	None	Vertical, occasionally not severe	Rarely	Moderately dilated	P u vei ph
33	M	65	None	57	Has about 1 fit a week, rarely goes 2 weeks without one	A few min.	Nocturnal, rarely in daytime	Often sees colored lights before a fit	Frontal not very marked	Occasionally	Moderately contracted	I
34	M	17	None	6	Has 3 or 4 fits a day, has had 20 fits in one day	About 2 min.	Both at daytime and at night	None, except perhaps a little weakness in legs	Frontal marked after a fit	None	Moderately dilated	Ph p u vei has city tal)
35	F	26	None	10	Has 3 or 4 fits a year, formerly they were more frequent	½ hour	Nocturnal	None, unless irritability	Vertical severe and frequent; occipital rarely	Frequent	Moderately dilated	Ph p u vei
36	F	32	None	30	Has 3 or 4 fits a month, formerly had 3 or 4 in a day	20 min.	Nocturnal, sometimes in the daytime	None	None, except occipital: rarely after a fit	None	Normal	P u l vei
37	M	16	None	15	Has 3 or 4 fits a week	A few min.	Day also nocturnal	A "giddiness in the head"	Frontal frequent	Matutinal frequent and bad	Normal	P u vei
38	F	35	None	24	Has 1 fit in a month, formerly many fits, and attacks of numbers of fits in sequence	15 min.	Nocturnal, rarely in the daytime	"Nervous feeling"	Frontal bad & frequent, also occipital	Constantly matutinal	Moderately dilated	P u vei
39	F	17	....	......	....	......	......	......	......	......	......	....
40	M	17	None	2	Has 1 or 2 fits a day, rarely has gone a month or so without an attack	5 min.	Both at daytime and at night	Strange feelings inside	Frontal and temporal	Occasionally	Normal	P u l vei tra phy
41	M	15	None	14	Has 2 fits a day	3 or 4 min.	Nocturnal	Rarely a feeling of unsteadiness	Frontal occasional but bad	2 or 3 times a day	Normal	Cent cup. ting
42	M	15	None	12	Has 1 fit a month	3 or 4 min.	Daytime	None, (unless "irritable")	Frontal not frequent or severe	Rare	Normal	
43	F	28	None	12	Has 6 or 7 fits during the day, and some attacks of 5 or 6 in rapid sequence	2 or 3 min.	Both at daytime and at night	Sometimes a slight pain in left hand	Occipital severe and frequent, vertical after a fit	None	Dilated	Phy pu vein

nd Refraction Mydriatic.	Mydriatic used.	Vision and Refraction after Mydriatic.	Description of Glasses Ordered.	Length of time Glasses were in use.	Condition since wearing Glasses.
$\frac{6}{18}$ n.i. $\frac{6}{24}$ .i.	G. A. S. grs. iiss. ad ℥ i. t. d. ut. 7 days	$<\frac{6}{60} + \begin{matrix}+1.75\text{ D.}\\+6\text{ D.}\end{matrix} = \frac{-1.75\text{ D.}}{+4\text{ D. cyl. ax.}\mid\text{v.}} = \frac{6}{12}$ $<\frac{6}{60} + \begin{matrix}+1.25\text{ D.}\\+6.50\text{ D.}\end{matrix} = \frac{-1.25\text{ D.}}{-4\text{ D. ax.}\mid\text{v.}} = \frac{6}{12}$	Ordered cylinders only for constant wear	11 mos.	June—No fits since glasses; much better. August—Ditto. September—One fit, was much excited about obtaining a situation
R. $\frac{6}{9}$ n.i. L. $\frac{6}{9}$ n.i.	Do. grs. iv. ad ℥ i.	$\frac{6}{24}$ + 1.75 D. = $\frac{6}{6}$ $\frac{6}{24}$ + 1.25 D. = $\frac{6}{6}$	Refused glasses + 1 D. for near work		
$\frac{6}{6}$ let n.i. $\frac{6}{9}$ let n.i.	Do.	$\frac{6}{6}$ iv. + 1 D. = $\frac{6}{6}$ $\frac{6}{6}$ iv. + 1 D. = $\frac{6}{6}$	No glasses		
R. $\frac{6}{6}$ n.i. L. $\frac{6}{6}$ n.i.	Do.	$\frac{6}{12}$ + 1.50 D. = $\frac{6}{6}$ let. $\frac{6}{12}$ let. + 1.50 D. = $\frac{6}{6}$ let.	No glasses		
H.m. 1 D. = $\frac{6}{6}$ Do.	Do.	$\frac{6}{12}$ + 1.25 D. = $\frac{6}{6}$ $\frac{6}{12}$ let. + 1.25 D. = $\frac{6}{6}$	No glasses		
H.m. 1.25 D. = $\frac{6}{6}$ let Do.	Homatropine	+ 1.50 D. = $\frac{6}{6}$ let. Do.	+ 1 D. for distance, + 5.50 D. for near work	10 mos.	June—No fits since glasses, though still sensations. Ceph. and vertigo are much better September—No fits, still has occasional sensations. No ceph. and vertigo is rare now
R. $\frac{6}{6}$ n.i. L. $\frac{6}{18}$ n.i.	Gutt. atrop. sulph. grs. iv., ad ℥i. t.d. ut. 3 days	+ 2 D. = $\frac{6}{6}$ + 1.25 D. = $\frac{6}{6}$	$\left.\begin{matrix}+1\text{ D.}\\+.50\text{ D.}\end{matrix}\right\}$ for near work	4 mos.	July—Is much better. September—No fits since July till 18th inst., when one took place
$\frac{6}{12}$ let n.i. $\frac{6}{9}$ let n.i.	Do.	$+\begin{matrix}+3.50\text{ D.}\\+5\text{ D.}\end{matrix}\frac{+3.50\text{ D.}}{+1.50\text{ D. cyl. ax.}\diagdown 60^\circ} = \frac{6}{6}$ $+\begin{matrix}+3\text{ D.}\\+4\text{ D.}\end{matrix}\frac{+3\text{ D.}}{+1.50\text{ D. cyl. ax.}\diagdown 160^\circ} = \frac{6}{6}$	Ordered less + 1 D. sph. for constant wear	10 mos.	Health is much better. Ceph. much improved. Has had 3 fits during this time
H.m. 1 D. = $\frac{6}{6}$ n.i.	Do.	$\frac{6}{24}$ + 2.25 D. = $\frac{6}{6}$ $\frac{6}{18}$ + 2.25 D. = $\frac{6}{6}$	+ 1 D. for constant wear	10 mos.	September — Has not worn glasses regularly. Fits many less in number. Ceph. better
R. $\frac{6}{6}$ L. $\frac{6}{6}$	Do.	$\left.\begin{matrix}\frac{6}{24}+2\text{ D.}=\frac{6}{6}\\\frac{6}{18}+2\text{ D.}=\frac{6}{6}\end{matrix}\right\} = \frac{6}{6}$ let.	+ 1 D.	6 mos.	June—Now has 1 fit a month. Ceph. better. Ordered to wear spectacles constantly
R. $\frac{6}{9}$ let L. $\frac{6}{6}$ ii.	Do.	$\frac{+.50\text{ D.}}{+2\text{ D. cyl. ax.}\mid\text{v.}} = \frac{6}{6}$ $\frac{+1\text{ D.}}{+1.50\text{ D. cyl. ax.}\diagup 55^\circ} = \frac{6}{6}$	Ordered less + 1 D. sph. for constant wear	—	Has not worn glasses and remains *in statu quo*
R. $\frac{6}{18}$ n.i. L. $\frac{6}{18}$ n.i.	Do.	$\frac{6}{12}\text{ i.} \times \begin{matrix}+.50\text{ D.}\\+1.75\text{ D.}\end{matrix}\frac{0}{+2.25\text{ D. cyl. ax.}\mid\text{v.}} = \frac{6}{6}$ let. $\frac{6}{12}\text{ i.} \times \begin{matrix}+2.25\text{ D.}\\.75\text{ D.}\end{matrix}\frac{0}{+3\text{ D. cyl. ax.}\mid\text{v.}} = \frac{6}{9}$ i.	Ordered c̄ — .50 D. sph. for constant wear		
R. $\frac{6}{6}$ let L. $\frac{6}{9}$ n.i.	Do.	$\frac{6}{12}$ + .50 D. = $\frac{6}{6}$ let. $\frac{6}{12}$ i. + .75 D. = $\frac{6}{6}$ let.	No glasses.		
H.m. .50 D. = $\frac{6}{6}$ let Do. $\frac{6}{6}$	Do.	$\frac{6}{36}$ + 1.50 D. = $\frac{6}{6}$ $\frac{6}{24}$ + 1.50 D. = $\frac{6}{6}$	No glasses.		
H.m. .50 D. = $\frac{6}{6}$ let Do. $\frac{6}{6}$	Do.	$\left.\begin{matrix}\frac{6}{36}+2\text{ D.}=\frac{6}{6}\\\frac{6}{24}+2\text{ D.}=\frac{6}{6}\end{matrix}\right\} = \frac{6}{6}$ let.	+ 1 D. for near work	9 mos.	July—Has not worn glasses regularly; says he is much better. September—Has worn glasses. Has had no fits
H.m. 1 D. = $\frac{6}{6}$ Do.	Do.	$\frac{6}{60}$ + 3.50 D. = $\frac{6}{6}$ iii. $<\frac{6}{60}$ + 3.50 D. = $\frac{6}{6}$	+ 2 D. for constant wear	9 mos.	March—Has had 1 mild fit since glasses. April—No fits. May—Six slight fits in sequence (frightened by cab). July—No fits. Ceph. rare now. September—Has had 4 slight fits in one morning. Mother says she is better in every way

No. of Case.	Sex.	Age.	Family History as to Fits.	Age when Fits began	Frequency, &c., of Fits.	Duration of Fits.	Time when Fits occur.	Aura.	Cephalalgia.	Vertigo.	Pupils.	Fundi
44	M	25	None	11	Has 1 fit a week	A few min.	Matutinal, has had 1 only at night	None	Vertical and parietal, severe and frequent	Frequent	Moderately dilated, slow reaction	Central cup., ting L. ant lens ha genital
45	M	18	None	15	Has 1 fit a month	20 min.	Daytime	Hissing in the ears about 5 minutes before a fit	None	Very rare	Normal	Puls veins
46	M	16	None	13	Has 2 or 3 fits every night	3 or 4 min.	Nocturnal	"Funny feelings in stomach, forehead and eyes"	None	Rare, sometimes before and after a fit	Moderately dilated	Puls veins. phys. c
47	M	31	None	24	Has 1 fit at variable periods, but many sensations, formerly many more, about 1 or more in 14 days	A few min.	Daytime	Singing in ears and giddiness	None	None, except just before a fit	Dilated	—
48	M	35	None	11 mos.	Daily, several, has gone 3 weeks without	3 min.	Nocturnal, seldom in day	Pain in right side of body and singing in the ears	Temporo-frontal constant	Rare	Moderately dilated, slow reaction	Discs l slight ting v L.E. phys. c
49	F	14	None	3	Has 1 fit a month, from 3 to 13 years of age, no fits	A few min.	Matutinal	None	Frontal frequent, but only severe after a fit	Occasionally very bad	Moderately dilated	Discs. l pulsa veins. small cup.
50	M	11	None	9	Has 2 fits a week, has gone 7 months without a fit	3 or 4 min.	Matutinal	His "eyes keep moving," and he sees "colored lights before a fit"	Frontal frequent	Rare	Dilated	Discs l slight ting vei
51	M	19	Has 3 sisters with fits, one of these died in a fit at 26 years of age, 2 sisters squint	14	Has 3 fits a week, formerly 1 fit daily	A few min.	Nocturnal	None	Frontal bad & frequent	Rare	Normal	Pulsa veins. int. s mus & n crescent
52	M	43	None	42	Has had many sensations, but only 3 fits	A few min.	Daytime	None	Occipital severe and frequent	None	Moderately contracted	Discs wl slight n crescent
53	F	53	None	12	Has fits at variable periods, now about 1 a month, used to have attacks of fits	A few min.	Nocturnal	None	Fronto-temporal very severe and constant, occipital occasionally	Frequent & severe	Normal	—
54	M	22	None	21	Has about 1 fit a month, occasionally 2 or 3 in rapid sequence	A few min.	Matutinal	None	None, occasionally frontal after a fit	Matutinal daily	Moderately dilated	Pulsa veins. phys. c
55	M	28	None	22	Has about 3 sensations a month, even more, about 2 bad fits a year	A few min.	Matutinal, rarely at night	None unless a twisting to his right side	Frontal, occasionally occipital, both less than formerly	None	Normal	—
56	F	17	Eldest brother has had 2 fits	2	Nearly daily, had no fits from 2 to 12 years of age	A few min.	Daytime	None	None	None	Moderately dilated	Pulsat veins. extr. str
57	M	28	None	9	Has 1 fit a fortnight	2 or 3 min.	Both in daytime and at night	A "rising to head, & then it twists round to the left"	Vertical, occasionally not marked	None, but has unsteady head feelings	Moderately dilated	Discs wh pulsat veins

nd Refraction e Mydriatic.	Mydriatic used.	Vision and Refraction after Mydriatic.	Description of Glasses Ordered.	Length of time Glasses worn in use.	Condition since wearing Glasses.
3 D. = $\frac{6}{12}$ -2.50 D. = $\frac{6}{12}$ ii.	Homatropine	-3 D. = $\frac{6}{12}$ -2.50 D. = $\frac{6}{12}$	$-$ 2.50 D. for constant wear	9 mos.	JULY—Has now about 1 fit in 3 months. Thinks they are stronger in character. AUGUST—About the same. SEPTEMBER—Has had 2 fits since last note
t. H.m. .50 D. = $\frac{6}{6}$ ii. st. H.m. 1 D. = $\frac{6}{6}$ ii.	G. A. S. grs. iv., ad ℥i. t.d. ut. 3 days	$+$ 1.50 D. = $\frac{6}{6}$ let. Do.	No glasses		
$\frac{6}{12}$ ii. n.i. $\frac{6}{12}$ iii. n.i.	Do. 7 days	$\frac{6}{18} + 2\text{ D.} = \frac{6}{12} + \frac{-2.50\text{ D.}}{+2.25\text{ D.}} = \frac{6}{6}$ $\frac{6}{60} + 2\text{ D.} = \frac{6}{12} + \frac{-4\text{ D.}}{+1.75\text{ D.}} = \frac{6}{6}$	$\frac{+1.25\text{ D.}}{-4.50\text{D. cyl. ax.} \diagup 170^\circ}$ [C.W. $\frac{+0.75\text{ D.}}{-5.50\text{D. cyl. ax.} \diagdown 170^\circ}$	6 mos.	JUNE—Remains *in statu quo*
3.50 D. = $\frac{6}{6}$ iv. 3 D. = $\frac{6}{6}$	Homatropine	-1 D. = $\frac{6}{6}$ let. -1.50 D = $\frac{6}{6}$	$-$ 1.50 D. $-$ 2 D. C.W.		Has not worn glasses
R. $\frac{6}{6}$ n.i. L. $\frac{6}{6}$ n.i.	Do.	$\frac{6}{18} + 1$ D. = $\frac{6}{6}$ $\frac{6}{12} + 1.50$ D. = $\frac{6}{6}$	No glasses		Remains about the same
R. $\frac{6}{12}$ n.i. L. $\frac{6}{18}$ n.i.	G. A. S. grs. iv., ad ℥i. t.d. ut. 3 days	$\frac{6}{36}$ i. $+ \frac{+2.50\text{ D.}}{+5\text{ D.}} = \frac{+2\text{ D.}}{+2.50\text{ D. cyl. ax.} \mid \text{v.}} = \frac{6}{6}$ $\frac{6}{36} + \frac{+1.50\text{ D.}}{+4.50\text{ D.}} = \frac{+1.50\text{ D.}}{+8\text{ D. cyl. ax.} \mid \text{v.}} = \frac{6}{12}$	Ordered less + 1 D. sph. for constant wear	6 mos.	JUNE—Has had 1 fit since glasses. SEPTEMBER—No fits (one in July?). Much better in health
R. $\frac{6}{6}$ let. L. $\frac{6}{6}$ let.	Do.	$+ \frac{+1\text{ D.}}{+1.50\text{ D.}} \frac{+1\text{ D. sph.}}{+.50\text{ D. cyl. ax.} \mid \text{v.}} = \frac{6}{6}$ let. $+ \frac{+1.25\text{ D.}}{+1.75\text{ D.}} \frac{+1.25\text{ D.}}{+.50\text{ D. cyl. ax.} \mid \text{v.}} = \frac{6}{6}$	No glasses		
nts fingers only	Do.	$+ \frac{+.50\text{ D.}}{+1.50\text{ D.}} = \frac{0}{+1\text{D. cyl. ax.} \mid \text{v.}} = \frac{6}{6}$ iv. $+ \frac{-12\text{ D.}}{-9\text{ D.}} \frac{-12\text{ D. sph.}}{} = \frac{6}{60}$	$\frac{-1\text{ D. sph.}}{+1\text{ D. cyl. ax.} \mid \text{v.}}$ C.W. $-$ 8 D.	8 mos.	JULY—Has only had 2 fits since glasses, which he cannot wear regularly. Is better. SEPTEMBER—Still continues better. ? 1 fit
$-$ 1.50 D. = $\frac{6}{6}$ $-$ 3 D. = $\frac{6}{6}$ iii.	Homatropine	$\frac{6}{18} - 1$ D. = $\frac{6}{6}$ let. $\frac{6}{36} - 2.25$ D. = $\frac{6}{6}$ let.	$-$ .75 D. $-$ 2 D. C.W.	8 mos.	SEPTEMBER—Has had no fits but occasional sensations
.m. 7 D. = $\frac{6}{6}$ let. .m. 8 D. = $\frac{6}{6}$ ii.	G. A. S. grs. iv., ad ℥i. t.d. ut. 3 days	$+$ 7.50 D. = $\frac{6}{6}$ $+$ 8 D. = $\frac{6}{6}$ let.	Ordered and refused.	—	Has not worn glasses
t.—1.50 D. = $\frac{6}{6}$ ii. —1.50 D. = $\frac{6}{6}$ iv.	Do.	$+ \frac{+.50\text{ D.}}{+2\text{ D.}} = \frac{0}{+1.50\text{ D. cyl. ax.} \mid \text{v.}} = \frac{6}{6}$ $+ \frac{+1.50\text{ D.}}{+.50\text{ D.}} \frac{0}{+1\text{ D. cyl. ax.} \mid \text{v.}} = \frac{6}{6}$	Ordered c̄ — .50 D. sph. for constant wear	8 mos.	JUNE—Has gone some weeks without fits, then had 3 at one time in sequence, and now has been 3 weeks without fit. JULY—No fits, no ceph. Vertigo much better. SEPTEMBER Do., do., do.
R. $\frac{6}{6}$ i. n.i. L. $\frac{6}{6}$ n.i.	Homatropine	$\frac{6}{18}$ n.i. $+ \frac{\text{em.}}{+1\text{ D.}} = \frac{0}{+1\text{ D. cyl. ax.} \mid \text{v.}} = \frac{6}{6}$ iv. $\frac{6}{18}$ iv. $+ 1$ D. = $\frac{6}{6}$ $+ \frac{+.50\text{ D.}}{+1.50\text{ D.}} \frac{+.50\text{ D.}}{+1\text{ D. cyl. ax.} \mid \text{v.}} = \frac{6}{6}$ iv.	Glasses refused.	—	Has not worn glasses
R. $\frac{6}{18}$ n.i. L. $\frac{6}{60}$ n.i.	G. A. S. grs. iv., t.d. ut. 3 days	$+ \frac{-1.50\text{ D.}}{+1\text{ D.}} \frac{+.25\text{ D.}}{-2.25\text{ D.} \mid 20^\circ} = \frac{6}{6}$ iii. $+ \frac{-1.75\text{ D.}}{+1.25\text{ D.}} \frac{0}{-2.25\text{ D. cyl. ax.} \diagdown 120^\circ} = \frac{6}{6}$ v.	$\frac{-.75\text{ D.}}{-2.25\text{ D. cyl.} \diagup 20^\circ}$ $\frac{-1\text{ D.}}{-2.25\text{ D. cyl.} \diagup 120^\circ}$	—	Has not worn glasses
R. $\frac{6}{18}$ n.i. L. $\frac{6}{18}$ n.i.	Do.	$+ \frac{-3\text{ D.}}{+3\text{ D.}} \frac{+3\text{ D.}}{-6\text{ D. cyl. ax. —}} = \frac{6}{12}$ $+ \frac{-3\text{ D.}}{+2.50\text{ D.}} \frac{+1.50\text{ D.}}{-6\text{ D. cyl. ax. —}} = \frac{6}{6}$	Ordered less + 1.50 D. sph. for constant wear	6 mos.	No fits while using atropine drops. JUNE—No fits. JULY—? 1 fit. Is much better in health

No. of Case.	Sex.	Age.	Family History as to Fits.	Age when Fits began	Frequency, &c., of Fits.	Duration of Fits.	Time when Fits occur.	Aura.	Cephalalgia.	Vertigo.	Pupils.	Fun
58	M	19	One brother had fits	18	Has about 1 fit in 3 weeks and sometimes attacks of many fits in rapid sequence	A few min.	Nocturnal and evening	Has post orbital pains before a fit	Frontal frequent and post-ocular as aura	Occasionally before a fit not marked	Dilated, slow reaction	Phys pul veins
59	F	32	None	7	Has about 1 fit a month, slight fits at 7 years, and decided fits at 11 years	10 min.	Matutinal	Twitching of right hand	Frontal and temporal frequent	None; unsteady feelings in head	Very dilated, slow reaction	Phys pul veins
60	F	36	Mother had fits at 62 years of age	19	Has about 1 fit a month	10 min.	Matutinal	Her arms twitch for some days before a fit	Frontal not aching, but dull heavy sensations	Rare	Moderately dilated	Discs hyper ting
61	F	14	None	11	Has about 6 fits in a day	About a min.	Nocturnal, also in daytime	Slight stiffness of mouth	Frontal severe	None	Dilated	Phys pul veins
62	M	16	None	13	Has 1 or more fits in a fortnight, when excited they are much more frequent	A few min.	Nocturnal, also in daytime	"Dazzling in eyes"	Frontal severe and frequent	Rare	Normal	
63	M	28	Grandfather had fits	20	Has 2 fits a month, formerly they were much more frequent	A few min.	Both in daytime and at night	Sensation in legs, has also "lightning flashes" before eyes	Fronto-temporal very severe and frequent	Rare, but has unsteady feelings in head often	Dilated	Discs hyper cup., ting
64	M	22	None	5	Very frequent, daily	5 min.	Nocturnal	None; has seen "spots" before his eyes	Frontal, but only marked after a fit	None	Moderately dilated	Phys, puls veins
65	F	32	None	30	At various periods, sometimes has 2 or three fits in rapid sequence	A few min.	Nocturnal	None	None	Frequent & severe	Moderately dilated	Norma neal n
66	F	52	None	23	At variable periods, occasionally with a 12 months' interval	A few min.	Daytime	Sees "hundreds and thousands of stars and colours"	Severe after a fit only; vertical	Frequent	Normal	Discs puls veins. phys.
67	F	20	None	4	Has often 8 fits a day. No fits between 4 and 7 years of age. Often attacks of several in rapid sequence	3 or 4 min.	Daytime, but occasionally nocturnal	"Lightning flashes and brilliant dots." Sensations in throat and eye	Fronto-temporal	None	Moderately dilated, slow reaction	No
68	M	21	None	17	Has 2 or 3 fits a day. Has gone 4 months without a fit	Under ½ hour	Nocturnal, rarely in daytime	Frontal pains	Frontal frequent	Just before a fit	Dilated, slow reaction	Myopic cents, ting v
69	F	42	None	42	Began 2 months ago. Has had 2 fits (separate)	A few min.	Daytime	None	Occipital constant, vertical seldom	Rare	Moderately contracted	Phys. puls veins
70	F	28	(1) One cousin, maternal; (2) one cousin, paternal; have fits	8	Has about 1 fit a week; no fits between 3 & 19 years of age. Sometimes 2 fits in 24 hours	10 min.	Daytime, rarely at night	None	Fronto-temporal, worse about time of fits	Rare, but has occasionally unsteady feelings	Moderately dilated	Discs hype puls veins, cup.
71	F	30	None	18	Daily; has laid 4 hours in succession of fits	A few min.	In daytime and at night	None	Frontal, worse after fits	Rarely	Moderately dilated	Discs mic, pu veins
72	F	13	None	10	Has 10 or 12 fits a day. Once had 114 fits in a week. Has also uncounted fits at night	2 min.	Do.	None	Frontal and occipital, but not at all marked	Do.	Normal	Puls. veins phys.
73	F	12	Father when quite young	5	Has attacks of many fits in rapid sequence, about every 3 months	Depending upon no. of fits	Do.	Clutching of the left hand, and beating of heart	Frontal frequent	Do	Do.	Optic very w atroph pings. ders ve in colo

Refraction ydriatic.	Mydriatic used.	Vision and Refraction after Mydriatic.	Description of Glasses Ordered.	Length of time Glasses were in use.	Condition since wearing Glasses.
· $\frac{6}{18}$ · $\frac{6}{12}$	G. A. S. grs. iv. ad ℥i. t. d. ut. 3 days	<$\frac{6}{60}$ — 1 D. = $\frac{6}{6}$ <$\frac{6}{36}$ — 1 D. = $\frac{6}{6}$	— 1.75 D. for constant wear	6 mos.	July—About the same. Aug.—1 slight fit since last here. September—No fits, no ceph., no vertigo
ot read.	Do., grs. iiss. t. d. do.	R. + + 1.50 D. + 1.75 D. L. Do.	No glasses		
$\frac{6}{6}$ let. $\frac{6}{6}$ let.	Homatropine	+ em. = — .50 D. = $\frac{6}{6}$ + 1.50 D. + 1 D. cyl. ax. | v. + em. — .50 D. = $\frac{6}{6}$ = $\frac{6}{6}$ + 2 D. + 1.50 D. cyl. ax. | v.	Ordered c̄ — .75 D. sph. for constant wear	6 mos.	Has not worn glasses
H.m. 1 D. = $\frac{6}{6}$ Do.	Nil.		+ 4.50 D. for near work		
$\frac{6}{6}$ let. $\frac{6}{6}$ let.	G. A. S. grs. iiss. ad ℥i. t. d. ut. 7 days	+ + 2.50 D. + 2.50 D. + 2.50 D. = $\frac{6}{6}$ Do. + 2.50 D. = $\frac{6}{6}$	+ 1.50 D. for constant wear	5 mos.	September—No fits, no ceph. Mother says he is quite well
n. .50 D = $\frac{6}{6}$ Do.	Homatropine	+ 1.50 D. = $\frac{6}{6}$ + 1.50 D. = $\frac{6}{6}$	+ 1 D. for near work	—	No note
iii. n.i. iv. n.i.	Do.	$\frac{6}{12}$ + .50 D. = $\frac{6}{6}$ $\frac{6}{6}$ + .50 D. = $\frac{6}{6}$	No glasses		
H.m. .50 D. $\frac{6}{6}$ let. Do.	G. A. S. grs. iv. ad t. d. ut. 3 days	$\frac{6}{12}$ let. + + .75 D. = + .75 D. = $\frac{6}{6}$ let. + 1.50 D. + .75 D. cyl. ax. | v. $\frac{6}{12}$ Do. Do.	Ordered for nearwork	5 mos.	September—Has had 1 fit since glasses, but his general condition is worse
no H.m. no H.m.	Do.	+ + 1.75 D. = + 1.75 D. = $\frac{6}{6}$ ii. + 2.25 D. + .50 D. cyl. ax. | v. + + 1.75 D. + 2.25 D. Do.	+ 1 D. for near work	Do.	June—No fits. September—3 weeks ago 3 fits. Does not wear glasses always
m. 1 D. = $\frac{6}{6}$ l.m. 1 D. = $\frac{6}{6}$	Homatropine	+ em. = + .75 D. = $\frac{6}{6}$ + 1.75 D. + 1.50 D. cyl. ax. | v. Do. Do.	Ordered less sph. for constant wear	4 mos.	July—2 fits. September—2 fits. Is better in health.
m. 1 D. = $\frac{6}{6}$ Do.	Do.	+ + 1.75 D. = + 1.75 D. = $\frac{6}{6}$ + 2.25 D. + .50 D. cyl. ax. | v. = $\frac{6}{6}$ + + 2.25 D. + 2.25 D. = $\frac{6}{6}$ + 3 D. + .75 D. cyl. ax. | v.	Ordered less + 1 D. sph.	—	Does not wear glasses
$\frac{6}{6}$ let. · $\frac{6}{6}$	G. A. S. grs. iiss. ad ℥i. t. d. ut. 3 days	+ + 1.50 D. + 1.75 D. + 1.50 D. sph. = $\frac{6}{6}$ Do. Do.	No glasses		
R. $\frac{6}{6}$ L. $\frac{6}{6}$	Do.	+ + 2.75 D. + 3.50 D. + + 2.25 D. + 2.75 D.	+ 2 D. sph. for constant wear	4 mos.	June—No fits while using atropine drops. July—Has had 2 fits and 3 slight sensations
$\frac{6}{6}$ let. $\frac{6}{6}$ let.	Do., grs. iv. ad ℥i. do.	$\frac{6}{12}$ iii. + + .25 D. = + .75 D. = $\frac{6}{6}$ iii. + .50 D. + .25 D. cyl. ax. | v. $\frac{6}{12}$ ii. Do. Do. = $\frac{6}{6}$ Do. cyl. ax. ⁄ 100°	+[illegible] D. sph. for near work	4 mos.	September — No fits since glasses, but still has sensations
not read.	Do., grs. iiss. a. d. ℥i. do.	+ em. = — .50 D. = good vision. + 1 D. + 1 D. cyl. ax. | v. + em. — .50 D. = good vision + 1.25 D. + 1.25 D. cyl. ax. ⁄ 120°	Ordered c̄ — .75 D. sph.	Do.	July—Mother says she is decidedly better. Fits are not so numerous. September—Better, but still has fits.
et. no H.m. o H.m.	Homatropine	+ .50 D. = $\frac{6}{6}$ let. + .75 D. = $\frac{6}{6}$ let.	No glasses		

No. of Case.	Sex.	Age.	Family History as to Fits.	Age when Fits began	Frequency, &c., of Fits.	Duration of Fits.	Time when Fits occur.	Aura.	Cephalalgia.	Vertigo.	Pupils.	Fu
90	F	8½	None ……	8½	Two days ago had 15 fits in rapid sequence	—	Daytime	None	None	None	Moderately dilated	Pu vei
91	F	19	None ……	19	Several fits at various periods, also many sensations	A few min.	Matutinal	None	Frontal frequent, occasional vertical	Rare	Normal	Disc hy pu vei
92	F	16	None ……	15	At variable periods, often many in rapid sequence; has gone a month without an attack	Do.	Daytime, but also at night	None	Vertical always & severe, temporal rare	Frequen	Do.	Pu vei
93	F	13	None ……	4	Has about 30 fits a day, also some during night not counted. Has had 150 in 1 week	Do.	In daytime and at night	"Strange feelings inside"	Frontal severe & frequent	None	Moderately dilated, slow reaction	
94	F	30	None ……	15	Has 2 or 3 fits daily	15 min.	Do.	None	Do.	Rare	Moderately dilated	
95	M	37	None ……	11	Has 2 or 3 fits daily, with 2 or 3 very heavy ones weekly	A moment	Nocturnal	Peculiar feelings in head, and giddiness	Fronto-vertical	Frequent & before fits	Normal	
96	M	40	None ……	36	Has 2 or 3 fits a day	Momentary	Do.	Peculiar sensations	Vertical and post-ocular, rarely occipital	Frequent	Do.	
97	F	24	None ……	12	Has about 1 fit a month; no fits between 12 and 21 years of age	10 min.	In daytime and at night	None	Frontal occasional & severe	Rare	Moderately dilated	R.E. cre L.E. phy Bot sati
98	F	27	One brother has fits	27	Has had 3 fits during the last 2 months, but has had many sensations	Do.	Daytime	Peculiar feelings in stomach	Frontal parietal for weeks together	Frequent, matutinal	Do.	Phy sligh ting
99	M	19	None ……	18	Has from 2 to 9 attacks daily since 8 months old	Momentary	In daytime, also at night	None	Frontal rare now, formerly very frequent	Rare, after a fit occasionally	Do.	Pul vein
100	M	16	One sister has fits	2	At variable periods, now 2 or more fits in a year	½ hour	—	None	Frontal severe and frequent, worse after a fit	Frequent & severe	Normal	Cent cup., ting

Refraction driatic.	Mydriatic used.	Vision and Refraction after Mydriatic.	Description of Glasses Ordered.	Length of time Glasses were in use.	Condition since wearing Glasses.
n.i. n.i.	G. A. S. grs. iiss. a. d. ℥i. t. d. ut. 7 days	$\frac{6}{36} + \begin{array}{l}+3.25\text{ D.} = \frac{6}{6}\\ +3\text{ D.}\end{array}$ $\frac{6}{60} + \begin{array}{l}+3\text{ D.}\\ +3.25\text{ D.} + 3\text{ D.} = \frac{6}{6}\end{array}$	+ 2 D. for constant wear	4 mos.	JULY—No fits. SEPTEMBER—No fits.
t. n.i. n.i.	Do., grs. iv. ad ℥i. do.	$\frac{6}{36} + 1.75\text{ D.} = \frac{6}{6}$ $\frac{6}{60}$ Do.	+ 1 D. for constant wear	Do.	SEPTEMBER — No fits. Ceph. much better, in fact rare
let.	Do.	$< \frac{6}{60} + \begin{array}{l}+2.25\text{ D.}\\ +3.50\text{ D.}\end{array} = \frac{+1.75\text{ D.} = \frac{6}{6}}{+1.25\text{ D. cyl. ax.} \mid \text{v.}}$ $< \frac{6}{60} + \begin{array}{l}+2.50\text{ D.}\\ +4\text{ D.}\end{array} = \frac{+1.75\text{ D.} = \frac{6}{6}}{+1.50\text{D.cyl.ax.}/120^\circ}$	Ordered less + 1 D. sph. for constant wear	Do.	JUNE—Had no fits since using atropine drops, which have apparently stopped a series of fits. JULY—1 slight fit, no vertigo. Ceph. better. SEPTEMBER—3 fits and sensations. Says she is very much better in health
$\frac{6}{12}$ $\frac{6}{12}$	Do., grs. iiss. ad ℥i. do.	$< \frac{6}{60} + \begin{array}{l}+2.50\text{ D.}\\ +5\text{ D.}\end{array} = \frac{+2.50\text{ D.} = \frac{6}{6}}{+2.50\text{ D. cyl. ax.} \mid \text{v.}}$ $< \frac{6}{60} + \begin{array}{l}+3.50\text{ D.}\\ +6\text{ D.}\end{array} \frac{+3.50\text{ D.} = \frac{6}{6}}{+2.50\text{ D. cyl. ax.} \mid \text{v.}}$	Do.	—	? if this case is hystero-epilepsy
n.i. n.i.	Homatropine	$+ \begin{array}{l}\text{em.}\\ -1.75\text{ D.}\end{array}$ $+ \begin{array}{l}+1\text{ D.}\\ +.50\text{ D.}\end{array}$			
2 D. = $\frac{6}{6}$ 2 D. = $\frac{6}{6}$	Do.	$+ \begin{array}{l}-1\text{ D.}\\ -2.25\text{ D.}\end{array} = \frac{-1.50\text{ D.} = \frac{6}{6}}{-1.25\text{ D. cyl. ax.}/100^\circ}$ $+ \begin{array}{l}-2\text{ D.}\\ -2\text{ D.}\end{array} \frac{-2\text{ D.} = \frac{6}{6}}{-.50\text{ D. cyl. ax.}/80^\circ}$	Ordered c̄ — .75 D. sph. for constant wear	—	No note
iii. iv.	G. A. S. grs. iv. ad ℥i. t. d. ut. 3 days	$\frac{6}{36} + \begin{array}{l}+2.50\text{ D.}\\ +1.75\text{ D.}\end{array} = \frac{+1.75\text{ D.} = \frac{6}{6}}{+.75\text{ D. cyl. ax.} -}$ $\frac{6}{60} + \begin{array}{l}+2\text{ D.}\\ +2.25\text{ D.}\end{array} = \frac{6}{6}$	Do. for near work (his work forbids the constant wear)	3 mos.	Cannot wear glasses at his work. Remains *in statu quo*
12 D. = $\frac{6}{18}$ ount fingers	Do.	$< \frac{6}{60} + \begin{array}{l}-12\text{ D.}\\ -10\text{ D.}\end{array} = \frac{-10\text{ D.} = \frac{6}{12}}{-2\text{ D. cyl. ax.} -}$ $+ \begin{array}{l}+2\text{ D.}\\ +3.50\text{ D.}\end{array}$ = sees no letters	Wears — 8 D. for constant wear		No note
let. let.	Do.	$\frac{6}{36} \frac{+.50\text{ D.} = \frac{6}{6}}{+.50\text{ D. cyl. ax.} / \text{v.}}$ $\frac{6}{24} \frac{+.50\text{ D.} = \frac{6}{6}}{+.25\text{ D. cyl. ax.}/\text{v.}}$	+ 1 D. for near work		
$\frac{6}{6}$ let.	Do.	$\frac{6}{12} + 1.25\text{ D.} = \frac{6}{6}$ $\frac{6}{6} + 1\text{ D.} = \frac{6}{6}$	No glasses		
n.i. n.i.	Do.	$+ \begin{array}{l}+1.50\text{ D.}\\ +1.25\text{ D.}\end{array} = \frac{+1.25\text{ D.} = \frac{6}{6}\text{ i.}}{+.25\text{ D. cyl. ax.} \mid \text{v.}}$ $+ \begin{array}{l}-.50\text{ D.}\\ +2.25\text{ D.}\end{array} \frac{+2.25\text{ D.} = \frac{6}{12}}{-2.50\text{ D. cyl. ax.}/20^\circ}$	Ordered less + 1 D. for constant wear	—	SEPTEMBER—Has not yet obtained glasses

Clinical Cases.

A CASE OF HYSTERIA IN A BOY, CHARACTERIZED BY REGULARLY RECURRING ATTACKS OF LETHARGY: TREATMENT BY HYPNOTISM.[1]

BY THEODORE DILLER, M.D., PITTSBURG, U.S.A.

THE valuable paper by Professor Hitzig, "On attacks of Lethargy and on Hypnotic Suggestion," published in *Brain* (Spring-Summer, 1893), has moved me to relate the prominent features in a highly interesting and instructive case of regularly recurring lethargy in a negro boy, aged 14 years. The chief phenomena manifested in my case were very similar to those observed by Professor Hitzig in his case. Furthermore, my patient was, like his, treated by hypnotism, but with less degree of success; indeed it is questionable whether any real lasting benefit resulted from the treatment in my case. The case is similar to Professor Hitzig's in that it was of traumatic origin (symptoms following a blow on the head); it was characterised by regularly recurring periods of sleep (eight days duration); there was lessened metabolism during the lethargic periods; the patient was highly susceptible to hypnotism.

I regret that I am unable to furnish exact data as to bodily weight of patient and as to his excretion of urea during the attacks and intervals.

The patient was seen by me for the first time at the Mercy Hospital, last November, at the request of Dr. Brumbaugh, who afterwards kindly transferred him to my care. At that time he was in one of his "spells." He was profoundly apathetic; face expressionless; eyes open. He could not be induced to talk or manifest the least interest in what was going on around him. A pinch or prick of the skin was unnoticed. My first impression was that it might be a case of stuporous insanity or of melancholia attonitia. But upon learning the history of the case and observing its subsequent developments, I became convinced that the phenomena manifested were those of hysteria.

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[1] Paper read before the Pittsburg Academy of Medicine, January 15, 1894.

History.—Charles Gibson, mulatto, aged 14. Up to the beginning of his present trouble he was regarded as an unusually bright and intelligent boy, but full of mischief and animal spirits. In 1889 he went to Washington, where he lived with a relative for three years, until September, 1892, when he returned to Pittsburg. Two years before his return here he met with an accident which marked the beginning of the disease from which he now suffers. He was engaged in a dispute with a companion, who, becoming angry, struck him on the head with a heavy iron rod, inflicting two wounds over the posterior part of the left parietal region, as witnessed by two scars close together, one-half and three-quarter inches long respectively. The boy, who was rendered unconscious by the blow, was carried to a place of retirement, where he recovered consciousness and was attended by a physician who sewed up and dressed the wounds. It was not thought at the time that a fracture had been produced. The lad apparently made a good recovery without sequelæ. But a few weeks after the injury he was taken with a spell of "mutism," preceded, however, for a few days by symptoms of giddiness. These spells occurred regularly once a month until a few weeks before I saw him, since which time they have occurred about every two weeks. The spells were characterised by complete apathy, and always lasted exactly eight days—never a longer or shorter period. During the attacks the boy could not be induced to talk or move, or in any way notice his surroundings. He passed his urine and fæces in bed. He would take no food, but drank some water during the attacks. Towards the end of the attacks the tongue and lips became dry, the teeth covered with sordes, and the breath very foul. The skin was cold, clammy, and the bodily weight evidently diminished. He states that he could remember much that was said to him while he was "under the spell." The beginning of the interval was always characterised by a returning desire for food, which, in a few days, amounted to a voracious appetite. For a few days he would eat an amount of food which would suffice for two able-bodied men.

During the intervals the boy was full of spirits, exceedingly bright and intelligent, up to all sorts of tricks and mischief. His cousin states he is "full of the devil."

Present State.—On November 30, 1892, I carefully examined the lad during one of his intervals, while he was at his best. I found him to be an unusually bright, intelligent boy, with excellent manners (such as might have put many white boys to shame). A search for morbid sensory and motor phenomena was entirely fruitless. An examination of the head revealed the presence of the scars already referred to. They were not painful. No depression in the skull could be discovered.

December 3, I hypnotized him by the suggestive method, as employed by Professor Bernheim, of Nancy, with the greatest ease. I suggested that the medicine I was giving him (a placebo) would be of great benefit to him.

December 5, hypnotized him in the presence of Drs. Koenig, Stewart, Barham, and McKennan. Arms were placed in a number of awkward and unusual positions when it was suggested that they would remain in those positions until removed by me. The arms always were left, until I moved them, in the positions I placed them, and often remained so for a length of time, impossible to be persevered in by any voluntary efforts. I suggested that he should fight, pray, cry, &c., all of which suggestions were obeyed by characteristic movements and attitudes of the hands and arms. Any part of the body was readily rendered anæsthetic by suggestions, so that pin pricks or other painful stimuli were unnoticed. These suggestions were repeated at several subsequent sittings. I then suggested that he would not have his next spell until after Christmas (the next attack was due a few days before Christmas). I did this, reasoning that it would be easier to effect by suggestion, a postponement of the attack than to entirely prevent its supervention. If I was able to effect a postponement of attack, then, I reasoned, this demonstration of power would, at a subsequent time, lend greater weight to a suggestion that the attacks should never return.

December 20, hypnotized; suggestion of December 5 renewed. (The next day I left the city, and remained absent until December 28).

December 23. It was noticed that he was restless, uneasy and wanted to be very industrious at work about the house (always, according to his cousin, a sure sign of an approaching attack).

24th. Loud, noisy, incoherent, calling people by wrong names.

25th. Apathetic; knew no one except his cousin.

26th and 27th. The same.

28th. I saw him. He was in a semi-stuporous condition, but knew me and called me by name. I administered an emetic of ipecac., which produced free emesis and seemed to waken him up somewhat.

30th. He called at my office, and, except that he was a little taciturn, seemed to be in his normal condition.

The duration of this attack was about five days; the shortest he had ever had. According to his cousin, it was less severe than any previous one, the lethargy being much less pronounced. The noisy prodroms were entirely new features. On the whole, the cousin pronounced the attack as very different from any he had previously had. As usual, he ate voraciously after this attack.

I again hypnotized him and suggested that he would never have another attack. I passed a very mild galvanic current through his head, from forehead to occiput, principally for its psychical effect. I usually repeated this at his subsequent visits.

January 9, 1893. Hypnotized and told he would never get another attack.

January 21. Called at office with cousin, in a semi-stupid condition. Hypnotized; suggested that he would be well on Monday (23rd).

January 23. Called at office with cousin, decidedly more stupid than at last visit. Movements sluggish; face expressionless; refuses to talk. Rather strong faradic current passed through back and shoulders, causing him to show lively discontent, as evidenced by movements and outcries. He had eaten nothing for two days—nor had he had a movement of the bowels during this period. I sent for a biscuit, which I forced him to eat under threat of applying the strong current to his back. I also administered a drop of croton oil.

January 24. Oil acted, producing several copious stools. Still very lethargic. Called at my office. Breath very offensive; sordes on teeth and lips. I forced him to eat a sandwich.

26th. Called at office alone; bright and talkative to-day; ate and drank freely.

It will be noted that this attack lasted five days. It was much less profound than usual. The boy, instead of remaining in bed throughout it, was, by my orders, kept up, and was compelled to eat twice, although the sum total of the food ingested was trifling.

On the whole both the attacks here described were shorter and less severe and characteristic than usual. The suggestion made before the first of these two attacks was a failure in that the attack came on before Christmas, when it was suggested that it would not come on till after Christmas. Yet its appearance was actually postponed a few days, and when it did come it was quite atypical, of shorter duration and less severe than usual. The suggestions made after the first of these attacks, that he would never have another spell, was largely a failure too; yet it resulted in making the attack which did come of three days less than the usual duration and by far the mildest he had ever had.

In both these attacks I noticed a lowered state of vital tone as shown in many ways. The skin was cold and clammy; there were sordes on tongue, lips and teeth; the breath was very offensive; there was an appreciable diminution in bodily weight; the pulse was slow and sluggish; the excretion of urine and fæces was markedly diminished.

I was greatly disappointed in not being able to continue my treatment and observations in the case, but I was prevented from doing so by a curious circumstance, which throws some light on the character of my little patient.

Soon after recovering from the second of the attacks described, he called at the police head-quarters in Pittsburg and told a pitiable story, relating that he had been lost in the city by his mother, who had travelled on to Washington. He told his tale so well that his story was readily credited, and he was furnished by the police department with a ticket to Washington. He then went home, secretly packed up a few effects and at once proceeded to Washington, and has since made his home with a relative in that city. The authorities at the police head-quarters were greatly astonished when they learned they had been duped, as they stated that the boy's earnestness of manner and the entire plausibility of his story had been such as to disarm suspicion in any one.

I suspect that the lad was moved to resort to this piece of stratagem through fear of further coercive measures in treatment. Moreover, he had heard some talk of an operation being performed on his head, and I have reason to believe that he dreaded this radical procedure would be executed.

The scars on the head were not painful. No hysterogenetic zones were discoverable. Yet the attacks of lethargy in themselves; the absence of all objective symptoms; the mannerisms and *morale* of the boy, together with his extreme susceptibility to hypnotism; and lastly (and of great significance) the marked modification in character and duration of the two attacks brought about by hypnotic suggestion, indicated that the attacks were hysterical. Moreover, the masterly treatise on "Hysteria," by Gilles de la Tourette, reviewed in a recent number of BRAIN, seem to conclusively show that these sleep phenomena belong to the manifestations of hysteria. This view is confirmed by Prof. Hitzig's opinion, and to it I would add the feeble weight of my own approval.

With these meagre and unsatisfactory report and remarks I will conclude what I have to say, leaving you, gentlemen, to make any further conclusions or deductions, or to take issue with those I have made.

A CASE OF LEFT HOMONYMOUS HEMIANOPSIA.

BY JOHN TURNER, M.B. (ABERD.)

Senior Assistant Medical Officer, Essex County Asylum.

EMMA JOHNSON, aged 54, a house-painter's wife, was admitted on July 5, 1889. She had a paralytic stroke four or five years ago. Duration of insanity said to be three weeks. First attack: Facts indicating insanity on the admission paper were :—Unable to say where she is or what she has been doing. Memory for recent events a blank. Bit the hand of a nurse and attempted to strike one of the other inmates of the union.

On admission.—Stout, dark haired woman, complexion rather dark. Loss of power in left arm and leg, and sensibility to pricking is diminished in the limbs of this side but equal for both sides of face. Is in a state of mania, restless and excited, especially when talked to. Says her name is "anything." That she has sold her rings to get bread with. That she lives on her rent. She has no money and her husband does no work. Good health.

July 8.—She is very helpless on account of the paralysis of left arm and leg. The arm is contractured and semiflexed at elbow, and flexed at wrist and fingers ; left leg very slightly flexed at knee and fixed so that she cannot move it (anchylosed). She can lift both her legs off the bed, but with difficulty. Both plantar reflexes are absent, both knee jerks much exaggerated. Tendency to ankle clonus in left foot. Sensibility of left leg to pricking is almost *nil*, and much blunted and perverted in the right. Left leg cold, right warm. Both legs are marked extensively from the knees inclusive to feet with cicatrices of ulcers. Has some white cicatrices on her arms, especially the right, and the skin is darkened around. Chest healthy. She is very delusive ; lies and talks to her daughter, who she says is up above her and swears a great deal. Said she walked a mile yesterday. Is in bed.

September 20.—Her pupils are unequal, the right being the larger, and remained so till she died. They do not react to light, and the right only accommodates for near objects. Tongue straight. When she smiles her mouth is drawn up on the left. *Analgesia:* Marked on both sides of face, chest, neck, arms, and legs. *Anæsthesia:* Some on the left side, none on the right upper extremity, none on either side of lower extremity that I can detect.

Localization of prick or touch.—Is generally seven or eight inches out on left side, less on the right (tested by telling her to place her fingers on the spot that I prick), same for legs and arms (tells correctly how many times she is touched or pricked on legs); *Mentally:* Is very talkative, boisterious spirits as a rule, delusion that her husband visits her every night. During 1890 no marked signs to note. As a rule is very cheerful and easily aroused to boisterous mirth. Sits quietly all day. Vomited one night in August.

March 20, 1891.—In a state of secondary dementia, dirty in habits and helpless. Grins and laughs when spoken to.

June 20.—She is stout and looking very well. Very talkative; will sit and talk to another patient who is not taking the slightest notice of her. Laughs frequently; excitable and irritable, and uses filthy language.

August 24.—She cannot walk without assistance. Protrudes tongue to left. She is quite unaware when I prick her on the left side of face or left hand, but calls out directly I do so on the right, or on either leg. She keeps chattering and laughing, but is very obstinate, and when asked to do anything replies as a rule "I shan't." Does not seem able to fix her attention on anything for long together. She does not appear deaf. She can see with either eye, will tell the time, grasp pennies held before her without any hesitation, and count them correctly. Always called the sovereign I was testing her with, half-a-sovereign. There is no doubt that the *left* half of each field of vision is blind, but not I think quite up to fixation line. It is impossible to test her with any great precision, as she will not fix her eyes on any object sufficiently long together, and when I held up a little piece of paper on the end of a pencil she took no notice of it, but she immediately grasped at a penny held on her right side, but took no notice of one on her left until it nearly approached the middle line.

August 28.—Tested again to-day for hemiopia. When she is talking to me and looking straight at me, if I hold up a half-crown on either side of her about three inches apart from one another, between her and me and equally far from the middle line, and ask her what she sees, she will reply "a shilling," and will put up her hand to catch hold of the one on her right. She does not perceive the other till I move it past fixation point, and then she grasps it without hesitation. Each eye was tested separately with similar results.

In September she was confined to bed with erysipelas of face, started probably from an injury—a blow to eye. Vomited several times during this illness. Her temperature rose to 104° F., but in the course of a few days fell to normal again.

In December she fell out of bed and fractured her left clavicle, but seems to suffer no pain from the injury or examination.

March 10, 1892.—She ate a quantity of soap this morning and her lips are much swollen. She has done this once before. Sits talking and laughing to herself and other patients.

May 18.—Is confined to bed, where she lies huddled up on her left side. Her left foot and lower part of leg is swollen and the instep is ulcerated.

June 3.—Tested for hemiopia again to-day with similar results. Slight superficial sore on left buttock; speaks and knows what I say.

June 10.—Abscess formed over sacrum which has burst.

June 21.—Enormous hard brawny swelling of right parotid. Superficial sore about size of five shillings on left hip.

June 24.—To-day she died.

Post-mortem five hours after death.

Body.—Thin, rigor mortis present. *Sores*: Slight superficial one on left hip, and some very slight superficial abraded spots about inner aspect of left knee, right hip and right foot. Hard swelling of right parotid.

Atrophy of left arm and leg; there was about an inch difference in the circumference of arm, forearm, and thigh; no shortening.

Head.—*Scalp* thick.

Skull of average thickness and density, deeply grooved for blood vessels internally, no deeply marked pacchionian depressions.

Dura-mater firmly adherent to skull over the posterior part of the superior longitudinal sinus, which latter contained a little dark clot easily removed.

Arachnoid.—A bath of lymph having a jelly-like aspect was seen to cover the whole vertex beneath the arachnoid. There were deeper lakelets over the posterior end of the two lower frontal convolutions and lower end of ascending frontal on either side, and also over each superior parietal lobe, but deeper on the right. The arachnoid was opaque and thickened. Meninges not adherent to cortex.

Circle of Willis abnormal, there did not appear to be any posterior connecting vessel on the right side. The vessels were generally small and slightly atheromatous.

Cerebrum.—There was atrophy of the whole cerebrum, but more pronounced on the right, especially in the right præ-frontal lobe and the tips of both temporal lobes. A portion of the second occipital convolution on the right had a shrivelled appearance contrasting very markedly with the similar convolution on the left (see fig. 1). Brain firm and tough.

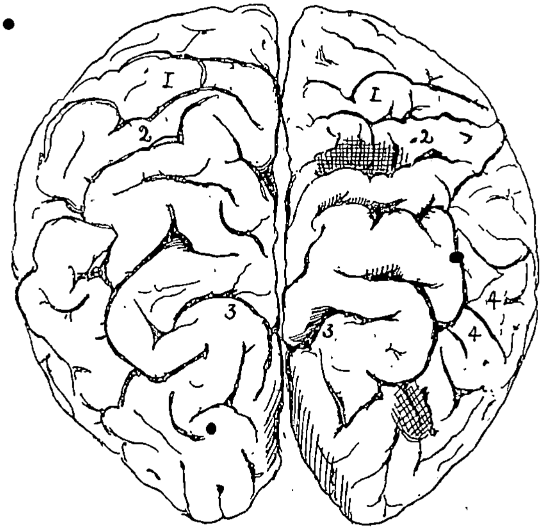


Fig. 1.

1. Is placed on ascending frontal convolution.
2. ,, ,, parietal ,,
3. Is adjacent to the parieto-occipital fissure.
4.4. Placed on angular gyrus.

The cross-shaded parts represent the seat of the lesion in the ascending parietal and adjacent superior parietal convolutions; and also the atrophied convolution in the second occipital gyrus.

The figure is outlined from a photograph taken of the brain.

Small cortical lesions in *right* hemisphere—old hæmorrhagic softenings.

(1) About the size of a pea in the back part of superior frontal.

(2) A rather large lesion implicating the upper part of ascending parietal and adjacent superior parietal lobe.

(3) A lesion in the second occipital convolution (see fig. 2). This lesion existed as a small irregular cyst situated at the junc-

tion of the cortex and white matter, and from it extended a degenerated tract running downwards and forwards beneath the posterior horn of the lateral ventricle for a distance of 6.5 centimetres. This tract occupied the lower most part of the optic radiations (see figs. 2 and 3).

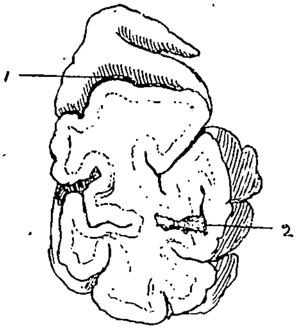


FIG. 2.

Horizontal section through parietal occipital fissure.
1. Parietal occipital fissure.
2. Small cyst.

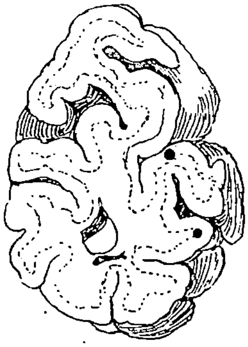


FIG. 3.

Horizontal section about 1 cm. in advance of fig. 2 shows the posterior horn of lateral ventricle with small patch of degenerated tissue (black).

Both figures are outlined from photographs.

Lateral ventricles enormously distended with fluid. Small granulations on the lining membranes at the anterior ends; softened patch (old hæmorrhagic focus) observed on surface of *left* caudate nucleus about an inch from anterior end; this was seen on the floor of the ventricle.

Both optic thalami appeared shrunken but the right was the more atrophied of the two.

Transverse vertical sections made at distance of one centimetre from each other revealed numerous softened patches in the white matter of each præfrontal lobe and for a large extent below the right ascending parietal and right superior parietal lobes. Small softened patches also in right lenticular nucleus. The extent of the lesion in the occipital lobe has been referred to.

Right crus cerebri smaller than left. In the hardened preparation there was a difference of three millimetres in greatest length.

Right half of pons flattened, and the motor tracts on right side much smaller than on the left.

Right pyramid of medulla oblongata smaller than left.

Floor of fourth ventricle studded with coarse granulations.

No lesions observed in cerebellum.

Weight of fresh brain :—

Right cerebral hemisphere (stript)	...	313	grammes.
Left ,, ,, ,,	...	340	,,
Arachnoid and Pia	...	14	,,
Pons and Corpora Quadrigemina	...	21	,,
Cerebellum	...	106	,,
Total	...	794	,,

Eleven and a half ounces (327 cc.) of fluid collected from base beneath membranes and from ventricles.

Thorax.—Small patch of pneumonia at base of right lung. Heart weighed 227 grammes.

Abdomen.—Liver 1297 grammes, right kidney 113 grammes, left kidney 99 grammes.

This case is of interest, inasmuch as it presents a strictly limited and small lesion of a portion of the cortex and white matter in a region of the brain concerning whose function considerable difference of opinion still exists.

Briefly summarized there was with the small occipital lesion well-marked and persistent left homonymous hemianopsia, although neither the cuneus or the angular gyrus were involved.

Unfortunately the mental condition of the patient was such that a more precise account of the visual defects could not be obtained; it was impossible to accurately define the limits of the hemianopsia, although there was little doubt but that it reached up to fixation point, nor was it possible to test her for color hemiopia, &c.

Reviews and Notices of Books.

Lead Poisoning in its Acute and Chronic forms. THOMAS OLIVER, M.A., M.D., F.R.C.P.

DR. Oliver has reproduced these very interesting lectures (Goulstonian, 1892) in book-form, thus contributing a valuable addition to the literature of this subject.

We confine ourselves in this notice to that part of his work which touches on the influence of lead on the nervous system. This, as may be expected, is manifested in a great variety of clinical forms. We will first notice the very serious acute nervous manifestation of plumbism known as lead encephalopathy.

After presenting the general symptoms of lead poisoning for a longer or shorter time, the patient passes into a condition easily taken for ordinary hysteria, so called "toxic hysteria"; this is soon followed by troubles of vision, amblyopia, temporary amaurosis. Convulsions, epileptoid in character, followed by fatal coma, may close the scene; or various mental troubles may supervene, such as mania or melancholia. There may be recovery with permanent or temporary loss of sight.

Dr. Oliver is of opinion that the severe pains of lead colic may be due to affection of the great sympathetic ganglia. This, however, must be purely hypothetical, from lack of facts either for or against, though the inequality of the pupils noted in so many cases is of interest in this connection.

A considerable space is devoted to the discussion of the chronic nervous symptoms of plumbism.

At the head of these come naturally the classic "wrist-drop." The occasional paralysis of the Duchenne-Erb group of muscles is also noticed.

Another form the chronic nervous affection may take, is one resembling progressive muscular atrophy of the Aran-Duchenne type and also of the leg or peroneal type.

An unusual form is described as "generalized" paralysis, in which large groups of muscles may be almost suddenly attacked; the disease may resemble an acute ascending paralysis with affection of limbs and trunk, but the head and neck are said to be usually spared. This form tends to recover, often rapidly. Death may occur from asphyxia.

The sensory symptoms of plumbism are not very striking. Dr. Oliver finds that pain often precedes the paralysis. Patches of anæsthesia and analgesia are fairly common on the arms and legs. Actual hyperæsthesia has been described.

The question as to whether the paralysis is of central or peripheral origin receives some attention. The myopathic theory was based on the assumption that the muscles contained the largest amount of lead. This assumption has been shown to be erroneous, and it has been found that the muscles, compared with other organs, contain the least quantity of lead, bulk for bulk.

The facts in support of the localisation of the seat of lesion in the grey matter of the cord is next discussed. V. Monakow found atrophy of the ganglion cells in the anterior lesions of the cervical cord. Oeller found capillary hæmorrhages, many of them involving the anterior cornual cells. The peripheral nerve trunks were also affected. Vulpian found myelitis after experimental lead poisoning.

These seem to be all the facts adduced in favour of the central hypothesis, and they seem to us too meagre to generalize from. The neuropathic theory of the causation of paralysis has scarcely received sufficient consideration in our opinion. The sensory symptoms mentioned as being common can scarcely be explained on any central hypothesis, and Dr. Oliver is careful to distinguish these symptoms from those of a hysterical nature.

Some interesting observations are made upon optic changes. He finds optic neuritis may be an early symptom in plumbism, and this quite apart from any kidney affection. There may be complete recovery from this, or the condition may progress to optic atrophy and total blindness.

Several interesting cases are quoted, with drawings of the discs, and pathological appearances after death.

Hysterical manifestations are occasionally met with, and may be prodromal, or may even be the only symptoms of the disease.

H. H. TOOTH.

Brain Surgery, by ALLEN STARR, New York, 1893, 8vo, pp. 288.

THIS work, written by a physician who, although not himself an operator, has seen much cerebral surgery, aims rather at providing a judicial summary of our present knowledge than at originality or great minuteness of detail, but such a summary will be of no small value to those who have not the time or opportunity to study the numerous scattered records of the last few years. The scope of the book, its pleasant style, and even its faults—occasional dogmatism upon points which are still *sub judice,* and the enunciation of propositions without reference to exceptions—are those of a work, which will appeal less to the specialist than to him who requires only a practical working knowledge of the subject. We can indeed hardly explain the general character of "Brain Surgery" better than by quoting the following paragraph, which sufficiently indicates Dr. Starr's brevity and lucidity, accompanied by dogmatism :—

"The speech areas are of four kinds and in four locations. They are limited to the left hemisphere in right-handed persons and to the right hemisphere in left-handed persons. There is *the motor speech area* in the posterior part of the third frontal convolution, in which the movements concerned in the act of speech are controlled. The use of language and the power of talking are affected when this region is destroyed. There is *the auditory speech area* in the first and second temporal convolutions, in which the memories of word sounds are stored up. The understanding of language and the power of recollecting the names of objects are lost when this region is destroyed. There is *the visual speech area* in the lower parietal region, in which the memories of printed words are stored up. The understanding of written language and the power to read are lost when this region is destroyed. The *power of writing* is a part of speech and is usually lost when the motor speech area is destroyed, but its exact location is not fully determined; some cases pointing to the second frontal convolution, others to the lower parietal convolution near the hand centre as its probable cortical position." It should be added that the above is all that we find in the book on the subject of speech.

The first chapter of "Brain Surgery" deals mainly with the outlines of diagnosis, topography, and other preliminary subjects. Hare's method is preferred for the delimitation of the fissure of Rolando, and Reid's for the Sylvian fissure; but here as elsewhere, we miss the descriptions of alternatives, and there is no reference to the work of Thane, Anderson and Makins, and others. It is, however, pointed out that great accuracy in the marking out of cerebral regions is not practically required, as the trephine opening is always necessarily sufficiently large to allow of some margin of error. The chapter concludes with the following, as the possible indications for operation:—"Operations may be performed for the relief of epilepsy, for the cure of imbecility, for the removal of clots, for the opening of abscesses, for the excision of tumours, for the relief of intracranial pressure—with or without drainage of the lateral ventricles—and for the cure of traumatic insanity." The usual traumatic conditions are not mentioned here or elsewhere in the book.

In the second chapter is an account of trephining for epilepsy, which deals mainly with post-traumatic conditions. The cases are divided into four groups, viz.:—1. Those in which the external evidences of injury and the focal symptoms point to the same region of the brain as being the seat of the lesion. 2. Those in which these do not coincide, it being then advised to operate in the region indicated by the nervous symptoms; two interesting cases are recorded to illustrate that the latter are a safer guide to the real site of the lesion than are the local signs. 3. Of operation in cases of general epilepsy, without focal symptoms, but of traumatic origin, the account is naturally most unfavourable; "occasionally the fits are relieved"; "the operation must be regarded as entirely exploratory" and "often nothing is found . . . and no result is obtained." This will be the experience of most surgeons, but more might well have been said upon the interesting question of the relief of epilepsy by all surgical procedures, *i.e.*, by operations *per se*, a relief which has given rise to so many vain hopes in the past. 4. Lastly, in cases of focal epilepsy in which no lesion is found after opening the meninges, the excision of the affected cortical area is not advised, chiefly upon the usual ground that such a procedure can only lead to further sclerotic changes. The calculated mortality after operations for epilepsy is taken from the most recent statistics to be seven per cent., and the general results are by no means favourable; of thirteen original cases only three are said to be cured, and these are reported only at intervals of two, three, and four months after the operation. And among the collected cases the longest period of immunity is eight months. "The question then arises, shall we continue to trephine any cases of traumatic or localised epilepsy? Judging by general results which are unfavourable, we should be inclined to stop. But when it is taken into consideration that in no case is it possible without an operation to determine the exact pathological condition present, and that a certain proportion of the pathological conditions are removable, it is evident that an operation, if not attended with danger, may be reasonably undertaken. Some patients have undoubtedly been cured.' To this summary we may add that a very full account is given of the changes found in several of the author's cases, and that Dr. Van Giesen contributes a careful description of their histological appearances.

The section upon "Imbecility due to Microcephalus" is very interesting at the present time, but deals with various congenital conditions besides that of microcephaly. The cases are divided into three main groups, the characters of which may, however, be combined, viz.: 1. Hemiplegia, with or without athetosis. 2. Mental defects of various grades. 3. Sensory defects of various types, especially deafness and varieties of visual defect. Epileptiform seizures are common in all the types, and the treatment of the organic epilepsies of childhood is also discussed in this place. It is pointed out that the clinical type depends rather upon the site than upon the pathological nature of the lesion, the latter including such diverse conditions as traumata at the time of birth, intra-uterine encephalitis and post-natal diseases of all kinds. The common factor in all of these affections is the production of sclerotic atrophy, and an analysis of 343 cases shows the following to be the lesions met with:—Porencephalus (132), sclerosis with disappearance of nerve elements (97), maldevelopment of the cerebral cells without gross defect (32), atrophy from embolism or thrombosis (23), meningo-encephalitis with adhesions, &c. (21), cysts external to the brain (14), hæmorrhages (18), hydrocephalus (5), and unilateral hydrocephalus (1). The primary origin of these various conditions, to which the writer has devoted much attention, is discussed, but without any fresh results being arrived at. As to the value of operation, it is clear that in the majority of the above lesions nothing is to be expected from trephining, but, in some cases of maldevelopment, it is suggested that "it is possible that anything which may stimulate latent powers of growth or may remove those influences which interfere with development, might result in improvement." It appears to us to be entirely problematical whether surgical intervention can produce these results. The actual statistics are, as might be expected, not good; thus 34 cases of craniotomy are analysed (Lannelongue's being excluded as wanting in precision of detail), and these yield 14 deaths—a much larger proportion than that of the cerebral operations upon adults; of the cases which survive the records are regarded as being too indefinite, or too recent to allow a satisfactory judgment as to the possibility of cure or great amelioration. It is remarkable that there is throughout the chapter only incidental references to the interesting and important question of premature synostosis, which is presumably one of "those influences which interfere with development," although the recent work of Bourneville and others proves conclusively that its importance has been enormously exaggerated.

The sections upon trephining for traumatic hæmorrhage contain nothing new to the surgeon; and operation is not advised in non-traumatic apoplexy. The cases of Championnière and Michaux are briefly related, together with an original case, in which operation revealed, not a clot, but a patch of softening. The main objection raised to this operation is the difficulty of diagnosis of the two latter conditions, but surely there are many cases in which no real difficulty is encountered in distinguishing hæmorrhage from embolism or thrombosis! No stress is laid upon what we take to be a far more serious difficulty, the radically defective condition of the cerebral vessels and of the patient's general health.

The chapter upon abscess of the brain contains an excellent summary of our present knowledge, but makes no attempt to add to this.

In connection with tumours of the brain, Starr estimates that only seven per cent. are removable (tubercular masses being included), and he summarises the prospects of operation in the following paragraphs:—

"In any case which presents the general symptoms of brain tumour, and in which, during the progress of the disease, such local symptoms appear as indicate that the situation of the tumour is in or near the vertex of the convexity of the brain, the operation of trephining is indicated.

"This operation is not to be undertaken hastily in any case, as it is important to try the effect of anti-syphilitic treatment in those patients who may have had syphilis, and to watch for other symptoms or for signs of multiple tumours in patients who may have had tuberculosis. But if mercury and iodide of potassium fail to relieve the patient within three months, or if during that time the symptoms rapidly increase, the operation is not to be postponed.

"The chances of success are greatest in hard encapsulated sarcomata and fibromata, and in these cases the history will show little variability in the symptoms during the progress of the disease. These tumours are usually on the surface and are easily removed. The chances of success are worth taking in glio-sarcomata and in soft infiltrating gliomata with or without cysts, as life may be prolonged by the operation and as several operations may be done successively if the tumour recurs. Such cases usually show much variability in the symptoms, as the tumours are vascular. The dangers are greater in their removal, partly from hæmorrhage, partly from inability to excise them entirely without great loss of brain tissue and consequent shock. It is not to be forgotten that these tumours are liable to recur. The chances of success are fairly good in cysts of the brain, provided the wall of the cyst is excised or the healing of the cyst from adhesion of its walls can be secured by permanent drainage. To empty a cyst and close the wound merely invites a refilling with fluid and is useless.

"Secondary carcinoma and sarcoma are not favourable (*sic*) for operation, as the chances of their recurrence are great, and the endurance of the patient is impaired by the primary disease."

To this long quotation we may add that among ninety-seven collected records of operations, there are forty-two in which the removal of the tumour was accomplished, and recovery ensued. Starr, in common with many recent writers, regards cerebral gummata as being generally amenable to medical treatment and as therefore not usually calling for operation, a view which our own experience certainly confirms. We miss in the chapter all reference to the puzzling cases in which focal symptoms are present but are misleading in their indications of the site of the tumour; and the very important question of the diagnosis of the *depth* of the tumour is dismissed with the remark that "the differentiation of cortical from sub-cortical tumours is still impossible," a remark which is certainly not universally applicable.

In a chapter upon trephining for hydrocephalus and for the relief of intracranial pressure, Keen's operation for tapping the ventricles is described, and seven cases are collected, of which the only one thoroughly successful is that of Broca, in which a hydrocephalic imbecile, with contracture of the right arm and epilepsy, was treated by drainage of the ventricles with very great improvement. The operation is recommended in such cases as being fairly safe and as likely to give a favourable result if the hydrocephalus be not secondary to other grave lesion.

On the subject of trephining for insanity, which has recently attracted a good deal of attention, the information is meagre. It is estimated that two per cent. of all cases are due to injury, and that in such cases relief may be looked for. A few cases of this nature are mentioned, and it is concluded that "the number . . . is too small and the operation has been performed at a period too far removed from the trauma to warrant any definite conclusions." "It seems to me that in general paresis, a disease which has an organic lesion, a diffuse chronic meningo-encephalitis, it is impossible for the operation of trephining to do any good whatever."

By the above summary and quotations we hope to have indicated the general scope of "Brain Surgery," which is certainly a welcome addition to the literature of the subject, and in conclusion we may remark that this very impartial and fairly judicial work leaves the strong impression, which must have been growing steadily among those who have occasion to operate frequently, that at present, at least, cerebral surgery has proved less valuable than it at one time appeared likely to become, and that, unless the future brings considerable advances in our knowledge of pathology and diagnosis, this section of our art is little likely to advance in popularity.

WILLIAM THORBURN.

Études de Chirurgie médullaire. By A. CHIPAULT. Paris, Alcan, 1893, large 8vo, pp. 403.

THIS work is the first of a series, which is expected to extend to four similar volumes, and which is to embrace a full discussion of all questions connected with the surgical treatment of diseases and injuries of the spinal cord. Coming from the French surgeon who has especially devoted himself to this subject it is of great interest; and it contains by far the most complete account of the surgery of the spinal cord hitherto published. The extent of the recent developments of this branch of operative surgery may be gathered from the fact that over 500 cases are referred to, although it must be admitted that in a few of these, the operation has concerned the spine, and not the spinal cord.

Passing over a short historical introduction, we come to a description of the various operative procedures which have been adopted; and we may note in passing that the operation of opening the medullary canal is always spoken of as "lamnectomy," as suggested by Lloyd, a nomenclature, which although etymologically correct, is not likely in this country to oust the term "laminectomy" now generally employed. For this purpose the central vertical incision is advised in all cases, and the bones are divided by rongeurs and forceps, and not by the trephine. A good deal of stress is laid upon the sub-periosteal method of resection, which is in the experience of the reviewer unnecessary, and it is stated that re-implantation of the bone after excision has not yet been practised; the latter has, however, been done in Manchester, but without any apparent benefit; and it would appear that the re-implantation of bone, either here or in the skull, is not of any great subsequent value, it being very doubtful whether the graft continues to live as bone. The author is opposed to the view of Prof, Horsley that the dura-mater should be opened in all cases, and his reasons for not doing so in many cases are certainly weighty. Much space is devoted to the question of suture of the pia-mater; but we think uselessly, as it is admitted that the operation is impracticable in cases of separation of the ends of the cord, and there is no reason to suppose that it would be of any service in cases of clean section; in fact, the disturbance involved, even in the careful hands of Dr. Chipault, would probably be productive of more injury than benefit.

In connection with the question of technique, a careful description is given of the author's method of attacking the anterior aspect of the medullary canal, by turning the cord aside, and thus removing tubercular granulations, and even chiselling away the protruding bodies of fractured vertebræ. Although much can be done by following up tubercular tissue to the front of the cord, and although the reviewer has frequently had occasion to do so, still we think that Dr. Chipault urges the matter too much; and that such an operation as the removal of the posterior edge of the body of a fractured vertebra through the medullary canal is not unlikely to be followed by considerable injury to the cord, from the necessary stretching thereof; nor, as a matter of fact, do the results attained by this method appear to have been more successful than those of lamnectomy as generally practised. Much of the section relating to operative procedures is taken up with the discussion of methods which, aiming only at the treatment of tubercular lesions of the spine without reference to the cord, have no special interest for the neurologist, and these, although interesting in themselves, may therefore be here passed over. Other tentative procedures, such as wiring the vertebræ, are also referred to, and the latter appears to us to receive rather too flattering a reception. Quincke's method of spinal paracentesis is described, but the recent work of Ziemssen on this subject is not mentioned. (Possibly it was published after the book had gone to press.)

Coming to the detailed consideration of the treatment of injuries of the cord, we find little that is new; it is at once admitted that traumata of the cauda equina are, par excellence, the cases in which we are to expect good results, and the views expressed by the reviewer in 1887 are unmodified, except that we are advised not to delay operation for more than four weeks in cases which do not spontaneously recover. Equally certain is the small success obtained in injuries of the cord itself, and the failure is attributed largely to the operation having been undertaken too late, or to its not having been sufficiently radical; it is concluded from pathological study of the date of degeneration of the injured cord that little is to be hoped for where laminectomy is performed after the third day. It appears to us that insufficient weight is laid upon the inability of the cord to recover from destructive lesions of any kind, and that no reform in procedure is likely to overcome this difficulty. In one point of pathology Dr. Chipault differs from the reviewer, viz., in his conclusion that the majority of complete fractures of the bodies are followed by persistent compression, but here we cannot entirely agree with him, although the discussion of the question would require too much space for the compass of a review. Basing his conclusions upon this view, the reduction of displacements is advised as a substitute for laminectomy in injuries of the cervical region. Finally, at the conclusion of the section, is raised the interesting, and as yet unsolved question, how far are the cases reported as cured to be considered permanent recoveries, or how far do late secondary changes supervene?

With regard to gun-shot wounds, stabs, and other similar special lesions of the cord, there are some interesting collections of cases, but it is as yet too early to attempt generalisations.

The treatment of tubercular affections is next carefully considered, and the pathology of paraplegia in spinal caries is well discussed, without, however, providing any convincing solution of what still remains a somewhat open question; the main interest of the section lies in the enforcement of the author's method of attacking the bone disease through the medullary canal, already referred to and first enunciated in the *Revue de Chirurgie* for 1891. The general summary is as follows: But few cases of paraplegia due to tuberculosis anterior to the medullary canal are suitable for operation; the results have been unsatisfactory, and operation should be reserved for certain rare conditions, viz., paraplegia from pressure of cold abscesses; from direct compression by granulations; and from perimeningitis which has become sclerosed. Successes have been obtained in other cases, but many of these would have recovered without operation.

Tumours are distinctly the most favourable field for spinal surgery, and here success has been great, most of these being extra-medullary, readily removable and non-recurrent; operation is altogether discouraged in the case of intra-medullary growths but on evidence which is as yet somewhat incomplete. Finally, a few rarer conditions which have led to laminectomy are discussed, but a remarkable omission is that of spina bifida occulta, which appears likely to give excellent results, and one case of which was reported by Mr. Jones, in the *British Medical Journal* for 1891.

The above remarks may appear to be of a critical, if not hypercritical nature, but we would not leave the reader with the impression that we do not regard this as an excellent work. It contains much careful reasoning, and has been prepared with unlimited industry by a master of the subject; and its study will be essential to those who wish to keep themselves abreast with this branch of surgery. There are numerous illustrations, which, although many of them are singularly inartistic, serve well the purpose for which they are intended; but it is remarkable that in two large coloured plates of laminectomies the incision represented is not that recommended by the author.

WILLIAM THORBURN.

Brain Diagrams on Slates. (DANIELSSEN & Co., Beaumont Street, London, W.)

MESSRS. DANIELSSEN & Co. have recently, at the suggestion of Dr. Whitwell, of Menston Asylum, made a set of engraved diagrams of the brain on slates for use in *post-mortem* and dissecting rooms. The diagrams are life size, twenty-five in number, and arranged on ten slates. The engraved outlines are filled in with white enamel, the Sylvian, Rolandic, and parieto-occipital fissures being, however, coloured red. Besides diagrams of the surfaces of the brain, pons, and cerebellum, there are numerous sections both vertical and horizontal, ranging from the frontal region to the medulla oblongata. On the whole the details are clear and distinct but in some slates the outlines are very thick and the general appearance rather confused by the amount of detail. The slates seem to be well suited for use in the *post-mortem* room where the site of any disease of the brain visible to the naked eye can be marked on the diagram in chalk at once by the pathologist even though his hands are soiled and wet. A rubbing can be taken from the diagram for addition to the *post-mortem* record, the site of the lesion being copied in ink or colour from the slate. In addition, however, to their use in the *post-mortem* room the slates seem well suited for use in the lecture theatre. The following complete list of the diagrams will show the thoroughness which has been aimed at in the preparation of these slates.

1.—Vertex. Base.

2.—Left lateral surface; right lateral surface.

3.—Inner surface of left hemisphere; inner surface of right hemisphere.

4 (after Dalton).—Horizontal section through anterior band and splenium of the corpus callosum; horizontal section through the anterior commissure.

5 (after Dalton).—Vertical section through the frontal lobes in front of the corpus callosum; vertical section through the corpus callosum, caudate and lenticular nuclei.

6 (after Dalton).—Vertical section through the corpus callosum, anterior pillars of the fornix and optic chiasma; vertical section through the corpus callosum, optic thalamus and crura cerebri.

7 (after Dalton).—Vertical section through the corpus callosum, and anterior pair of tubercula quadrigemina; vertical section through the lobulus quadratus and angular convolution; vertical section through the lobulus quadratus, cuneus and ends of the lateral ventricles.

8 (after Alexander Bruce).—Transverse section of the pons and cerebellum at the level of the sixth nerve; transverse section through the pons varolii at the level of the exit of the fifth nerve.

9 (after Alexander Bruce).—Transverse section of the medulla oblongata at the point where the direct cerebellar tract passes backwards to the restiform body; transverse section of the pons varolii immediately above the level of middle cerebellar peduncles; transverse section through the posterior corpora quadrigemina.

10.—Pons inferior view; pons superior view; pons lateral view; cerebellum superior surface; cerebellum inferior surface.

R. PERCY SMITH.

Abstracts of British and Foreign Journals.

Abstract of papers upon unusual forms of Muscular Atrophy, in several members of one family. By J. A. Ormerod, M.D.

I. "Sur la Névrite interstitielle, hypertrophique, et progressive de l'enfance." Par MM. J. Dejerine et J. Sottar. (*Mémoires de la Société de Biologie*, série ix., tome v.)

The authors first draw attention to the form of muscular atrophy described by Charcot and Marie, Howard Tooth, and others, which is sometimes known as the "peroneal" or "leg" type of muscular atrophy, since it commences in the lower limbs, sometimes by the name proposed for it by Hoffman, viz., "progressive *neuritic* muscular atrophy," since it is thought by him to be caused by disease of the nerves rather than by disease of the muscle substance or of the spinal cord.

The present two cases, while resembling this form of muscular atrophy in certain important particulars, viz., in the primary localization and the course of the muscular atrophy, and in the existence of a family predisposition to the disease, yet present additional symptoms which, even during life, should mark them as distinct; and the elaborate investigation into the morbid anatomy of one case gives to the authors' observations an independent and additional value.

The cases are those of a brother and sister. Little of importance in the family history; father died at 45 from congestion of the lungs; mother alive and well, age 61 (her father was a drunkard and she was a natural child); nine children born to them; of whom five died young; while of the survivors two males are healthy, the remaining two are the patients under consideration.

Case 1 (authors' summary).—Muscular atrophy and sensory affection in a woman of 44. Onset in early life with deformity of the feet, for which tenotomy was practised at age of 6. Marked double club-foot (varus). Atrophy of leg and thigh muscles, most marked at the periphery. Atrophy of upper limbs (Aran-Duchenne type) diminishing from below upwards. Excessive spinal curvature (kypho-scoliosis). Well marked sensory trouble, delay in transmission of sensation, diminishing from periphery to centre. Lightning pains. Incoordination distinct in upper limbs, less distinct in the lower limbs on account of the atrophy. Walking difficult and uncertain, but still possible. Romberg's symptom present. Slight choreiform movements of head and trunk. Patellar reflex, olecranon reflex, and plantar reflex absent. Pupils small, with very sluggish reflex to light. Nystagmus (dynamic). Fibrillary contractions of muscles of limbs and face. Sphincters and nutrition of skin not affected. Marked alteration of electrical contractility without reaction of degeneration.

Autopsy.—Hypertrophy of nerve trunks and spinal nerve roots, with alteration of the posterior columns of the cord. Histological examination—atrophy of the primitive fasciculus of the muscles, with slight steatosis and well marked sclerosis of vessels without any interstitial myositis. Hypertrophic interstitial neuritis of the nerve trunks diminishing slightly from below upwards, highly advanced in the muscular and cutaneous nerves. In the nerve roots the interstitial lesion, being less fully organised, exhibits the character of a primitive mono-tubular, interstitial neuritis. Similar lesions in the spinal ganglia. In the cord—lumbar region, sclerosis of Goll's columns and Burdach's columns; cervical region, Goll's columns highly sclerosed in their posterior part, columns of Burdach comparatively sound. No bacilli found in the nerves.

Case 1.—Fanny H. Age 45 at time of death. A small, cachectic woman, who had never suffered from any illness except the present paralysis. This began in early life. She began to walk at 3 years of age, and that badly; her feet were noticed to turn then. The spinal curvature did not exist then, but developed gradually later. Tenotomy was done at the age of 12; apparently she walked worse after it. She married at 32, and has now one healthy child (examined by the author) age 11, and has had three miscarriages. The weakness of the legs has gradually increased, and during the last fifteen years the affection has spread to the arms.

Condition at time of observation (1890).—In the lower limbs there is extreme muscular atrophy of legs and thighs, diminishing towards the root of the limbs. The feet exhibit much deformity, thus there is extreme varus, so that the patient walks on the scaphoid; exaggeration of the plantar arch, dorsiflexion of the first phalanges of the toes, plantar flexion of the two last. No movements of the toes remain, and of the feet only a certain amount of abduction.

In the upper limbs there is marked muscular atrophy of the hands (thenar and interossei muscles), somewhat less of the forearms. Movements of the fingers remain, but are enfeebled. Muscles of shoulder and back badly developed.

Extreme spinal curvature, kypho-scoliosis, convexity to left, in upper part of spine.

Walking is only possible when she has her surgical boots on; without them indeed she can scarcely stand. There is a certain brusqueness of movement in the legs, but no pronounced ataxia. She can scarcely stand without supports. Romberg's sign is present in slight degree. The movements of the upper limbs exhibit a slight inco-ordination increased by closing the eyes. Tendon reactions, patellar and olecranon, absent; skin reflex from sole absent. No genito-urinary trouble.

As to sensation. In lower limbs, tactile sensibility is absent from the feet to the upper third of both legs, reappearing gradually above this level. Sensibility to pain is present, but in lower part of limbs transmission is delayed several seconds. Thermal sensibility is considerably impaired, and delayed in transmission. In upper limbs, sensibility to touch, temperature and pain is also affected. Sensibility is normal in face and trunk.

Lightning pains in left thigh for many years.

Slight "dynamic" nystagmus.

Re-examined in 1891 (November), when matters had got worse, with the following result:—

She cannot walk, and can only stand with support, and then loses equilibrium when the eyes are closed. There is some slight degree of paralysis in the lower part of the face as evidenced by a prominence and downward droop of the lower lip. Fibrillary contractions are clearly seen in the levators of the angles of the mouth, and also in the muscles of the shoulders and arms.

Eyes.—The right eye has been operated on for cataract. The left pupil is small, it contracts very slowly and imperfectly under light. Nystagmus when the eyes are moved to the side, none during rest. Fundus normal.

Inco-ordination of movement cannot be demonstrated in the lower limbs on account of the advanced paralysis, but in the upper limbs it is evident enough (having increased since the last observation), and it is intensified by closing the eyes. As regards muscular sense, she can, when the eyes are closed, imitate with one arm any position in which the other has been placed, but cannot at once touch with her finger any part of her body that she is told to.

Electrical conditions.—Farado-contractility extinct in muscles of legs, thighs and hands, diminished in forearms. Galvano-contractility extinct or diminished in same degree as farado-contractility. No alteration of polar formula. Much diminution of electro-sensibility.

Cutaneous sensibility had undergone no change since the last observation.

Mentally the patient was somewhat feeble and emotional, though not so distinctly affected as her brother.

General condition bad: she had bronchitis and emphysema. She died in January, 1892, outside the hospital, and was buried; but the authors in their zeal obtained leave to exhume the body, and owing to the emaciation of the subject and the extreme coldness of the weather, it was found in good preservation.

The nerves of the cauda equina, both anterior and posterior roots, were found to be much enlarged (double their usual thickness), greyish in colour, smooth. The nerve roots of a higher level (dorsal and cervical) were slightly, but not strikingly, enlarged. The cord itself was normal, except that the posterior columns appeared rather smaller than usual, and somewhat reddish grey in colour.

The muscles of the lower limbs were much atrophied and of a reddish grey, in other parts salmon-coloured, yellow, or streaky and variegated. The most affected were the muscles of the feet and the muscles of the legs. In the leg muscles, the lower parts, viz., those nearest the tendon, were generally more degenerated than the upper.

The anterior tibial nerve was normal above the anterior tarsal ligament, underneath the ligament thinned and shiny, and below it swollen. The posterior tibial, and two plantar nerves were greatly swollen, and gelatinous in appearance. In the upper limbs the muscles of the forearms were thinned but otherwise normal. The intrinsic muscles of the hands, however, were highly atrophied. The median nerve was enlarged, firm, and gelatinous in appearance. The ulnar and radial nerves were much less affected.

An elaborate microscopical examination was made of the peripheral nerves, nerve-trunks, nerve-roots, spinal cord, and muscles. In the muscles (L. anterior tibial, L. flexor hallucis brevis, R. thenar muscles) it was difficult to find any muscular tissues proper. A few strings of sarcous elements, enclosed in a copiously nucleated sarcolemma were seen, and also empty sheaths of sarcolemma; these were embedded in fatty tissue through which ran veins and arteries (the arterial coats being highly thickened) and degenerated nerves. There was no increase of connective tissue in the muscles.

In the cutaneous peripheral nerves of the leg no myeline remained, the nerve being reduced to a string of connective tissue; in those of the thigh, a few myeline tubes could be seen enwrapped in connective tissue. There appeared to be no Wallerian degeneration, but the myeline sheath had been gradually thinned and destroyed, and the axis cylinder in its turn exposed and destroyed, by the formation of connective tissue around the nervous parenchyma; this process beginning at the periphery of the limb and spreading upwards. The changes in the peripheral nerves of the muscles were exactly similar.

In the nerve-trunks were similar changes which might also be said, broadly speaking, to begin at the terminal parts and to spread with decreasing intensity up towards the roots. Thus in sections of the internal plantar nerve, there is some thickening of the epineurium, and very great thickening of the perineurium so that each nerve-fasciculus is surrounded by a thick ring of connective tissue; the endoneurium is also thickened, though in a less degree. In the nervous tubes themselves the myeline and the axis cylinder have disappeared, and are replaced by multi-nucleated connective tissue. In the nerves of the leg are similar changes, but the connective tissue is looser and less fibroid. In the sciatic trunk here and there a myeline sheath appears and within these (where visible) a normal axis cylinder.

Concerning the nerves of the upper limbs it need only be said that they showed the same lesions, but in a less advanced degree than those of the lower limbs.

A special description is given of the microscopical anatomy of the hypertrophied nerve-roots, as here the lesion was not so far advanced as to obscure its mode of development. The anterior roots were more highly diseased than the posterior. Each nerve tube was wrapped in a casing of loose newly-formed connective tissue composed of fusiform cells mostly parallel to the nervous axis. The myeline sheath was thinned in various degrees down to complete disappearance [but in no part broken up into blocks and balls, nor were the nuclei and protoplasm of the sarcolemma increased as in Wallerian degeneration], the axis cylinder was normal so long as there was any myeline left around it, and even in some parts where it lay bare. Transverse sections showed connective tissue massed around areas which represented nerve tubes, and which in some cases showed remains of myeline, in others myeline and granular *débris*, the results of nerve disintegration.

The lower spinal roots were more diseased than the upper.

The authors regard the process as an interstitial neuritis, a proliferation of the connective tissue around the individual nerve fibres, commencing at the periphery of the nerves and spreading along their trunks up to the nerve roots. This connective tissue increase causes, in its early stages, enlargement of the structures as seen in the hypertrophy of the nerve roots, while in its later stages of organization and contraction it destroys by compression the nervous parenchyma, first the myeline sheath, then the axis cylinder ("neuritis interstitielle hypertrophique monotubulaire à marche ascendante").

Bacteriological examination of the nerve tissue threw no further light on the nature of the disease.

As regards the cord, the main feature was a degeneration of the posterior columns; which would appear from its distribution to be secondary to the lesions of the lower nerve roots.

At the second sacral segment, Burdach's columns were degenerated along the edge of the posterior cornua of each side. Lissauer's tract was degenerated, the posterior cornua somewhat thinned.

At the second lumbar segment, the degeneration of Burdach's column was more diffuse, and began to shift towards the middle line.

At the twelfth dorsal segment, the area of degeneration borders the middle line, extending at its posterior part only into the column of Burdach.

At the fifth dorsal segment, the posterior third of Goll's column is degenerated, Burdach's column showing only a slight deficiency of fibres here and there. This degeneration of the posterior part of Goll's column extended right up the cord.

A certain amount of disease was noticed in Clarke's columns. The cells of the anterior cornua differed but little from the normal, such alteration as there was being quite trivial in proportion to the disease of the nerve roots. The nerve fibres in their course through the cord to the roots were perfectly normal, the proliferation of connective tissue beginning immediately the roots left the cord. The degeneration within the cord appears to have been an ordinary secondary sclerosis; the neuroglia of the cord as a whole was not affected. A ganglion from a posterior nerve root was examined. The proliferation of connective tissue involved it as it did the nerves, but had not progressed sufficiently to destroy the cells.

Case 2 (authors' summary).—Man, aged 34. Muscular atrophy associated with sensory affections and lightning pains, onset at 14 years of age. Commencement of atrophy in the leg muscles, later extension of it to the muscles of the hands. Muscles of the proximal parts of the limbs intact. Club feet (equinus). Atrophy of hand-muscles of the Aran-Duchenne type. Fibrillary contractions in the muscles of limbs and of face. Considerable alteration of faradic and galvanic contractility, but no reaction of degeneration. Curvature of spine (kypho-scoliosis). Ataxia of upper and lower limbs, inability to walk in the dark. Giving way of the legs. Romberg's sign. Nystagmus (dynamic). Pupils small, and of Argyll-Robertson type. Commencing atrophy of left optic disc. Deafness. Abolition of tendon reactions (at knee and elbow), and of skin reflex from sole. Considerable modification of the various modes of sensibility, diminishing from below upwards. Marked hypertrophy and induration of all such nerves of the limbs as are accessible to palpation. Severe lightning pains, sometimes causing the patient to fall down. Micturition, defæcation, and genital functions normal. No trophic lesions of skin. Syphilis at 24 years of age. Excess in alcohol. Mental degeneration.

Case 2.—Brother of last patient, age 34.

History.—Curvature of the spine had been noticed by his mother from the age of 4 or 5. While being treated for this with Sayre's jacket, at the age of 14, his paralytic symptoms began. He often fell when running, had some difficulty in using his hands. Writing became difficult; he had also to give up his work as compositor. His speech became peculiar. He had lightning pains in the limbs, particularly the lower limbs.

Unsteadiness of gait, especially when he first got up to walk, continued till the age of 21. He then fractured the right thigh (accidentally) twice running; and after this the motor troubles in the limbs increased so much that he had to give up work altogether.

Syphilis at the age of 21; some excess in alcohol from the age of 20 onwards.

Present state (February, 1890).—A somewhat short, anæmic man, with muscular atrophy of upper and lower limbs, and deformity of hands and feet, and some shortening of the right lower limb (due to the old fracture of the thigh).

Talipes of both feet, marked equinus with a slight degree of varus, plantar arch much exaggerated, length of foot shortened, first phalanx of toes (especially the great toe) in dorsiflexion, the second and third in plantar flexion. Well marked atrophy of the thenar muscles, less of the interossei. Much atrophy of the leg muscles, particularly of the antero-external group. The tibialis anticus can still produce a slight dorsiflexion of the ankle and the calf muscles a little plantar flexion. Little movement is possible beyond this. The thigh muscles are not exactly atrophied, but are badly developed. Nevertheless, they appear tolerably strong. There are fibrillary contractions of the muscles.

He can only stand with the help of a stick and with his surgical boots on. Romberg's sign is well marked. He walks somewhat on his heels, and with a slight amount of jactitation; he does not walk straight, but swerving as if from cerebellar disease.

Muscular atrophy (of the Aran-Duchenne type) in both hands, involving the thenar and hypothenar muscles, and the interossei; the thenar muscles are completely paralysed, the interossei retain a small amount of power. Fibrillary contractions in the abductor longus pollicis (L.). Muscles of forearms poorly developed; epitrochlear group atrophied. Flexors of fingers and of hands enfeebled: the other forearm muscles retain their power. No definite atrophy of arm and shoulder muscles; those of neck, face, larynx, pharynx, palate and tongue are normal. The deformity of the spine consists in a kypho-scoliosis, concavity to left, in the mid-dorsal region.

Sensibility.—In lower limbs tactile sensibility is abolished from feet to knees; at knees it reappears, but with errors in localization and delay in transmission, and gradually improves up the thigh. Sensibility to pain and to heat and cold also altered, the localization thereof imperfect, and the transmission delayed. In upper limbs there are similar modifications of sensibility, but less marked. In the trunk sensibility to touch and pain diminished. On face and within mouth sensibility normal in all its modes. Muscular sense normal.

Tendon reactions at patella, and at elbow, absent. Plantar reflex not obtained.

In moving the hands towards a given object, slight swervings are made; these are much more marked when the eyes are shut. Similar inco-ordination is seen in the movement of the legs, but chiefly when the eyes are closed. Vision normal. Pupils slightly unequal, rather small in size, no reaction to light, normal reaction to accommodation. Nystagmus when the eyes are moved, but not while they are at rest. Lightning pains in the lower limbs, worst when he walks, often so severe that he has to stop, or may even fall down. Speech slightly scanning. Though of fair intelligence, the patient shows some moral signs of mental degeneration.

An electrical examination (made February 17, 1890) showed that the atrophied muscles did not respond to faradism, though it appears that they reacted to a magneto-electric machine; to galvanism the leg muscles responded not at all, while the flexors of the forearm responded to 20 ma., but without alteration of polar formula.

Between the date of these first observations (1890), and the present time (March, 1893), there has been some alteration.

The muscular atrophy and the strength of the muscles remain much as before. The inco-ordination has increased, he cannot walk in the dusk; his legs often give way, sometimes without cause, sometimes when the lightning pains attack him. He can reproduce with one arm the attitude of the other, when his eyes are shut; but when told to touch a point of his body has to grope for it.

There are fibrillary contractions in the levators of the angle of the mouth, some drooping of the lower lip, and some difficulty of whistling.

A formerly existing deafness has steadily got worse.

An important point (specially examined for in view of the *post-mortem* data obtained from Case 1) is that all the nerves which can be felt—ulnar at elbow, musculo-spiral in its groove, median on inner aspect of arm, peroneal near head of fibula—are enlarged to double their usual size and very hard to touch. They are not, however, tender.

The type of disease to which clinically these cases appear at first sight to be most closely related, is (as stated at the commencement of the article) muscular atrophy of the peroneal type. So far, indeed, as the muscular atrophy is concerned the resemblance is very close indeed. In members of one family, at a comparatively early age, a chronic muscular atrophy begins in the legs, causing (or at least accompanied by) club-feet, and subsequently it spreads to the hands. The authors notice a small difference, viz., that in these cases the toes assume a claw-like position (dorsiflexion of the first phalanx, plantar flexion of the other phalanges), which we presume was due to a preponderance of atrophy in the interossei. More manifest differences, however, are seen when we consider other aspects of the cases than their muscular atrophy. Thus, on the sensory side, there is marked affection of sensation in all its modes, whereas in peroneal atrophy affection of sensation is the exception; and as regards that rather vague function, the muscular sense, it is noted that, though the patients could (with eyes shut) reproduce with one arm the position in which the other was placed, yet when told to touch a given part of their limbs, toe, finger, elbow, &c. (the eyes being shut), they could only do so by fumbling and groping. Again, as to co-ordination, the patients were ataxic. This fact (so far as the gait was concerned) was in the first case masked by the paralysis; but in the second there were the following symptoms of ataxy, viz., jactitation of the legs in walking, swerving and lurching, inability to stand with the eyes shut (Romberg's symptom), or to walk in the dark, difficulty in turning or in beginning to walk, sudden giving way of the legs. In the upper limbs the ataxia showed itself by clumsiness in picking up objects, and erroneous projection of the finger in touching a point, errors which were increased by closing the eyes. Along with this ataxia existed two important symptoms whereby the disease is assimilated to typical locomotor ataxy rather than to muscular atrophy, viz., (1) lightning pains, (2) the condition of the pupils, which exhibited miosis and (in the second case at least) reflex irido-phlegia. Lastly (and in view of the relationship of the patients, the fact is particularly interesting), there were some symptoms which are more usual in Friedreich's ataxia than in ordinary tabes or in muscular atrophy, to wit, (1) nystagmus, (2) spinal curvature, (3) the peculiar deformity of the feet.

Clinically, then, the disease appears to be a sort of hybrid, in which appear the symptoms of peroneal muscular atrophy, of true tabes, and of Friedreich's disease. All the more interesting, therefore, is the account of the morbid anatomy. Of this we have already given a full abstract; but it is to be remarked that, in spite of the symptoms pointing to spinal disease, no spinal lesion was found beyond such as could be reasonably ascribed to secondary degeneration of the posterior nerve roots (degeneration of postero-lateral and of Lissaner's columns, degeneration of postero-median columns higher up), while the primary lesion consisted partly in a simple muscular atrophy, partly, and more particularly, in an hypertrophic interstitial neuritis, starting from the very periphery of the nerves and spreading right up to their roots.

The authors are able to quote but one similar case, viz., that published by MM. Gombault and Mallet under the title, "Un cas de tabes ayant débuté dans l'enfance" (*Arch. de Médecine Experimentale*, 1889, p. 385). In this case also there was muscular atrophy, affection of sensibility, inco-ordination of movement, onset in infancy, and a similar hypertrophic neuritis of nerve and nerve roots, with posterior spinal sclerosis was found *post-mortem*. It was regarded, however, as a case of tabes in early life with special complications.

The suggestion with which the paper concludes is worth quotation, viz., that in similar obscure cases of muscular atrophy, we should examine by palpation such nerves as are accessible to us. In the second case enlargement of the nerves was thus detected during life.

II.—"Ueber chronische spinale Muskelatrophie in Kindesalter auf familiärer Basis." Von Prof. J. HOFFMANN (Heidelberg). *Deutsche Zeitschrift für Nervenheilkunde.*

Some prefatory remarks are made upon the present classification of muscular atrophies, which divides them, according to the supposed primary seat of the lesion, into spinal, neural, and muscular types. This classification is based, partly upon anatomical, partly upon clinical data. Among the clinical characteristics, heredity has hitherto been considered to appertain only to the muscular type (*e.g.*, the progressive muscular dystrophy of Erb), or to the neural type (peroneal atrophy). But the following cases shew that a progressive muscular atrophy of spinal origin may also exhibit this character, attacking, as do the other two types, several members of the same family.

Case 1.—A girl of 4 years old. Up to $\frac{3}{4}$ of a year nothing abnormal had been noticed, except that she was very fat; she could sit up and move both her arms. Soon after this she gradually became unable to stand, sit in bed, or even turn over in bed though she still moved the feet and arms. The subcutaneous fat began to disappear, and revealed wasting of the muscles.

She was examined in the spring of 1891.

The limbs and trunk were wasted, the face pale, but not wasted. No sign of rickets, thyroid not to be felt, no demonstrable enlargement of thymus. There was paresis and wasting of many of the muscles of the neck and shoulders, of the upper arms, of the forearms; the intrinsic muscles of the hands were thin and flabby, though their motor power was more or less retained. The tendon reactions were absent, the mechanical irritability of the muscles diminished; the nerve trunks were not thickened nor tender. No fibrillary twitchings. Electrical reaction of degeneration demonstrated in the biceps.

In the trunk, much weakness of the muscles of back and of abdomen. Lordosis of the lumbo-sacral region.

In the lower limbs, the muscles of buttocks, flexors of thighs, and all the thigh muscles were much atrophied and palsied. Muscles of legs weak and atrophied. Movements of toes slow, but possible. Tendon reactions quite absent. Plantar reflex weak. No fibrillary twitchings.

The paralysis was throughout of the flaccid type, symmetrical on the two sides; there was nothing like hypertrophy or lipomatosis of muscle; no trophic changes; sensibility appeared to be normal. And, except for the signs of bronchitis, there was no other token of disease. The chest trouble increased during the winter, and the child died therefrom in March, 1892.

Post-mortem.—The body was much wasted, and there was very little rigor mortis. The spinal cord was small at the level of the lumbar enlargement, but otherwise normal. The anterior nerve roots, however, were highly atrophied.

The muscular degeneration was most strikingly seen in the quadriceps femoris, yet many other muscles were of a pale, yellowish red, instead of their normal colour. Much more subcutaneous fat remained than was expected.

Microscopical examination gave the following results: Cerebrum, cerebellum, pons, and medulla normal, excepting the lower sections of the medulla where the degeneration of motor cells, more evident in the spinal cord, began to shew itself.

Spinal Cord.—In sections from the upper cervical segments, there were only a few (at most five or six) normal cells in the anterior cornua, the rest were atrophic, their processes being small and scanty, their bodies small, their nucleoli indistinct. No disease of blood vessels nor proliferation of neuroglia. The anterior nerve-roots, external to the cord, shewed some signs of degeneration.

In the lateral and part of the anterior columns (apparently the pyramidal tracts), and in the parts of the lateral column between the pyramidal tracts and the anterior cornua—was a slight increase of neuroglia. The spinal accessory bundle was degenerated.

At the third cervical segment, the number of sound and of diseased anterior cornual cells was about equal.

In the cervical enlargement (which was normal in size) there were very few sound cells in the anterior cornua (only four or five in about thirty sections), there were many cell spaces empty of contents. As before, there was degeneration of the anterior nerve roots outside the cord, and slight degeneration of them just within the pia mater. In the dorsal cord, similar changes but less striking. The alteration in the pyramidal tracts could be traced down to this level, though faintly.

The lumbar cord was shrunken; the degeneration of the anterior cornual cells was more marked here than at any other part. The anterior roots were degenerated almost up to the anterior horn; the pyramidal tracts were normal.

The disease of the anterior cornual cells (and of their nerve roots) thus extended through the whole length of the cord; it was symmetrical on both sides, and appeared to be degenerative, not inflammatory, in origin; since the neuroglia of the cornua, and their fine nerve network, was apparently normal.

Some bundles of the cauda equina were thin and pale.

Of the *nerves* that were examined, the anterior crural nerve shewed degeneration (nuclei increased in number, fibres broken up); so in a less degree did the peroneal nerve, still less the median and radial. The hypoglossal was normal. (This disease of the nerve trunks was less than would have been expected from the condition of the nerve roots.)

The changes in the *muscular tissue* were most marked in the quadriceps cruris. Here there was little muscle tissue left; the space that should have been occupied by a fibre was filled with aggregation of nuclei; while around the spaces lay the usual web of connective tissue. There was no fatty infiltration of this connective tissue. There were some muscle fibres visible in varying stages of atrophy, circular, not polygonal, in outline. There were hardly any hypertrophied muscle fibres; and no dichotomy, vacuolation, nor fatty degeneration of the muscle fibres. In the intramuscular nerves there were a considerable number of degenerating fibres.

Other muscles, *e.g.* (1) the muscles of the back, (2) the anterior tibial group, the deltoid and biceps brachii, (3) the intrinsic muscles of the hand, shewed degeneration, but in diminishing degree. The masseter and digastric, diaphragm, and cardiac muscles were normal.

Case 2.—Younger brother of Case 1. A boy of 2¾ years old at the time of first examination (November, 1891). Till three quarters of a year old he had been healthy, and had moved his limbs naturally, and could sit up. But he never learnt to stand or walk, became unable to sit up in his cradle, became strikingly fat, at the same time that he became weak and flabby.

A large, pale child; much fat in the subcutaneous tissue; the muscles beneath it wasted and flabby; he cannot lift his head from the pillow, nor sit up unless propped.

There was paresis of the muscles of the neck and shoulder girdle (muscular atrophy was difficult to demonstrate in consequence of the fatness); in the arms, weakness and wasting of the flexor group, paresis of the triceps; in the forearms, wasting chiefly of the flexor group; in the hands, a certain amount of weakness and wasting of the intrinsic muscles.

Weakness of abdominal, weakness and wasting of back muscles. No lordosis.

The most marked paralysis was in the glutei and muscles of thigh, flexion of the hip was impossible; abduction barely possible; extension of the knee very imperfect. Much weakness and wasting of leg muscles, though some movements of the toes and ankles could still be made.

As in the preceding case, the muscular atrophy was diffuse, though masked by subcutaneous fat; the paralysis was flaccid in type, the tendon reactions were absent, there were no fibrillary tremors; electrically reaction of degeneration was found. There were no sensory abnormalities and no trophic disorders of the skin, and in all other respects than the muscular atrophy of the body and limbs, the child was healthy.

The child got gradually worse, a feeble cough developed itself, the subcutaneous fat diminished as the muscular atrophy progressed. (The observations are recorded till August of 1892, *i.e.*, for nearly a year.)

The family history of these patients stands as follows:—

Neither in the condition of the parents, nor in the history of their parents, was there anything appearing to bear on the origin of the disease.

Fifteen children had been born to their parents.

(1) Boy, healthy at first, but early in life he lost the use of arms and legs. Was very fat. Died at 11 months.

(2) Girl, died of diphtheria when nearly three years old; not paralysed.

(3) Boy, died at one month, of fits; was very stout, but no paralysis developed.

(4) Girl, when $\frac{3}{4}$-year old could stand, but subsequently lost power of standing. The paralysis, according to the parents, began in the trunk, and was accompanied by pain.

(5) Boy, now 11 years old and healthy.

(6) Boy, very fat; paralysis began in the trunk; had pains.

(7) Girl, died at 11 days, of fits.

(8) Boy, a seven months child, died an hour after birth.

(9) Boy, suffered from obesity and from the paralysis, also from pains.

(10) Girl, Case 1.

(11) Boy, died of fits, aged 1½ weeks.

(12) Boy, Case 2.

(13) Boy, died of fits, aged 3½ weeks.

(14) Boy, died of diarrhœa, aged three months.

(15) Baby, a month old, so far healthy.

Out of this large family, therefore, six may be disregarded, as not having reached the age at which the disease might be expected to declare itself; and of the remaining nine, six (four boys and two girls) had the disease; one (a boy) had what appeared to be an early symptom, while only two (one boy, one girl) escaped it.

The mother gave it as her experience that in those who died of the disease no *rigor mortis* appeared. She regarded the occurrence of obesity as a bad omen. Towards the end of the first year, and often after the children had learnt to walk and stand, the paralysis began in the trunk and legs, spreading afterwards to the arms.

By a curious coincidence two other cases from a second family came under the notice of the author about the same time.

Case 3.—Boy, age ¾ when first seen (Nov. 1891). A bright little child, exhibiting no sign of rickets or of other disease than that here described. The limbs had much subcutaneous fat, but their muscles were small and flabby.

There was weakness of the muscles of the head and neck, so that he could barely turn his head on the pillow or lift it up unaided. Almost complete paralysis of the extensors of the spine, abdominal muscles very weak. Paralysis and atrophy of the glutei and flexors of the hip; very little power in the muscles of the thigh, rather more in those of the leg. In the upper limbs, paresis of muscles of shoulder girdle and of upper arms; even the hand muscles were small and weak. No fibrillation, no rigidity, tendon reactions absent; reaction of degeneration, complete in the thigh-muscles, partial in the legs and upper limbs. Sensibility normal.

Early in 1892 persistent cough began, which became gradually feebler, and blocking of the air-passages commenced. The paralysis of the arms increased. The child died of bronchitis, March 29, 1892. No *post-mortem* was made.

This child had been healthy till five months old, able to move its limbs and to sit up well. About that age it had a prolonged shivering fit, about 14 days after which the mother noticed that it moved its limbs less easily than before; definite paralysis of the legs was made out some eight weeks later.

Case 4.—A boy (apparently not seen by the author), elder brother of Case 3.

He had been large and sturdy from birth; got his teeth early, could say some sentences and sing at a year old. But in the course of his first year he became gradually and unaccountably weak in the back and neck; never learnt to walk, and later began to lose power in his arms. He died of bronchitis æt. 1¾.

He had been seen when 14 months old by Professor Schultze, of Bonn, who had found paralysis with reaction of degeneration in the quadriceps crudis and tibialis anticus of both sides.

The history of this second family is as follows:—

Parents healthy in every way—they had six children.

(1) Boy, Case 4.
2. Boy, present age, 8 years.
3. Boy, ,, ,, 7 ,,
4. Girl, ,, ,, 5 ,, } all healthy.
5. Girl, ,, ,, 3 ,,
(6) Boy, Case 3.

Upon these cases the author bases a general sketch of the disease.

Beyond the fact that it appears to run in families, nothing is known of the causation of the disease. The parents, ancestry, and unaffected brothers and sisters appear to be on the whole healthy. So do the victims of the disease during the first few months of their life. Usually in the second six months of life the onset takes place, in a subacute or still more gradual fashion. The natural kicking movements of the legs become weak and hampered, presumably from affection of the muscles of the hip and thigh, and the muscles of the back become weak; so that, if the child has already learnt to stand or sit up, it loses the power of so doing. Thus it remains for a variable time. Then paralysis begins in the muscles of the head and neck, shoulder and upper arm; it can barely turn its head in bed, and has difficulty in lifting its arms. The muscles of the leg (below the knee) are involved about the same time, and the weakness spreads through in less degree to those of the forearm and hand. With this paralysis is associated atrophy of the affected muscles. At the first this may be hidden, from the eye at any rate, by an abnormal amount of fat in the subcutaneous connective tissue; but this may disappear, revealing the muscular atrophy in all its severity.

There are neither cerebral nor bulbar symptoms, nor are the sphincters affected; there is no sensory affection; pains appear to be the exception. The paralysis is symmetrical, and progresses; so that death usually results before the fourth year of life from secondary pulmonary affections.

The order in which the muscles are affected appears to be (1) Muscles of back, of buttocks, flexors of hip, thigh muscles. (2) Muscles of head and neck, of shoulder-girdle and upper arm, of leg. (3) Intrinsic muscles of hands, and probably of toes.

The muscles are not rigid, but lax, and waste progressively; the tendon reactions are abolished; there is electrical reaction of degeneration; there are no fibrillary or fascicular twitchings. The muscles are not tender to touch, the nerve trunks are not tender nor enlarged. There is no hypertrophy of the muscles.

Post-mortem there is found atrophy or disappearance of the bulk of the anterior cornual cells throughout the whole cord, but most marked in the cervical and lumbar enlargements, and particularly in the lumbar. Atrophy of the anterior nerve-roots (at least externally to the cord), degeneration of the nerves (motor or mixed), and atrophy of the muscles supplied by them. The muscular atrophy is characterised by increase of the nuclei, the muscle tissue disappearing by degrees, without losing its striation, and without such changes as dichotomy of the fibre, hypertrophy of the fibre, vacuolation, or fatty infiltration. The affection of the nerves and muscles is to be regarded as secondary to the disease of the anterior cornua.

The diagnosis has to be made from infantile paralysis, (spinal and cerebral), peroneal muscular atrophy, progressive muscular dystrophy of Erb (pseudo-hypertrophic paralysis, infantile and juvenile muscular atrophy), the hereditary muscular atrophy of Leyden and Möbivs. It is distinct from all these diseases, inasmuch as it consists in a chronic degeneration of the anterior cornual cells, but, unlike most varieties of this disease (chronic anterior poliomyelitis, amyotrophic lateral sclerosis, progressive muscular atrophy of Aran-Duchenne type), it is a disease of infancy and runs in families.

The author has been able to find only one set of similar cases, viz., those published by Werdnig in the *Archiv. für Psychiatrie,* vol. xxii., p. 437. The abstractor wishes to draw attention here to "a case of progressive muscular atrophy in a child, with a spinal lesion," published by Drs. Thomson and Bruce in the *Edinburgh Hospital Reports*, vol. 1. This was an isolated case, there being no other cases in the family.

III.—"Ueber eine hereditäre Form der progressiven spinalen mit Bulbär-paralyse complicirten Muskel-atrophie." Von Professor Dr. M. BERNHARDT. (*Archiv. für pathologische Anatomie und Physiologie und für Klinische Medicin. Band* 115, *Hft.* 2, *p.* 197.

Case 1.—P., a large, somewhat powerful-built man, aged 40 (January, 1882), complaining of inability to use the right hand properly in writing. He occasionally also stumbled with the right leg.

In February he noticed a sort of choking sensation in the throat whenever he laughed. The speech had now become somewhat thick, low, and occasionally hurried. The tongue appeared normal, also the lips, face, ocular and pupillary movements.

September, 1882. Well-marked bulbar paralysis. Speech low, toneless, difficult to understand. Tongue partially atrophic, cannot be protruded beyond the teeth, its farado-contractility diminished. Labials badly pronounced; difficulty in holding a cigar in the lips. Fibrillary twitchings in muscles of lips and chin. Farado-contractility of these muscles normal. Chewing and swallowing difficult. Cannot now write at all. Has a peculiar hopping movement in going up stairs. Well-marked patellae tendon reflex.

The bulbar paralysis continued to increase in April and May, 1883, and the patient died in August of that year.

Case 2 (first cousin once removed to Case 1, L. being grandson to P.'s aunt). October, 1882. L., a man about 32 years of age, who complained of weakness of the arms and neck. His head had a peculiar pose, falling forward as a rule with the chin upon the chest; the patient could only lift it either by his hand, or by a swinging movement executed with his whole body. There was atrophy and paralysis of the neck and shoulder-muscles, to wit, the trapezii, sternomastoids, levators anguli scapulæ, supra-spinati, and deltoids. Movements of the forearms and fingers were possible, but enfeebled. Faradic contractility was diminished in the muscles of the arm, forearm and hand, and was barely perceptible in the muscles of the neck and shoulder. No reaction of degeneration could be made out with the constant current.

The speech, face, tongue, lower limbs and sphincters were not affected. Tendon reactions normal.

Half a year later the only additional symptom was an occasional difficulty in swallowing.

The patient then passed into other hands, but the author heard from his sister that long before his death (which took place in 1884, two years after he was first seen) he could not feed himself, and the physician who last attended him regarded the case as one of bulbar paralysis.

Case 3 (first cousin to Case 2).—B., a man of 36 or 37, seen once only, March 1, 1888.

Much mental and bodily exertion during the last two years. Three-quarters of a year ago a sensation began of tension and weakness in the muscles of the neck and throat, particularly on the left side, radiating towards the left shoulder. Paræsthesiæ in the left cheek, difficulty and pain in chewing food, trouble in swallowing, spasmodic cough at night, with some spasm of the glottis. Then difficulty in balancing his head, which would fall forward or to the side.

On examination the patient was thin generally, but there was marked atrophy of the trapezii, splenii, supra- and infra-spinati, deltoids; with great diminution of their farado-contractility, but no reaction of degeneration. Muscles of the arms, forearms and hands thin, but not distinctly atrophied. Movements of arms at shoulder-joint deficient.

The head could only be moved feebly and tended to sink forward, chin upon chest.

No fibrillary tremors.

Dyspnœa upon the least exertion.

Tongue could be moved, but was thin, pointed, and showed furrows of atrophy. Swallowing difficult, singing impossible.

Soft palate moved normally; lips moved normally, but speech was difficult.

Upper facial muscles normal.

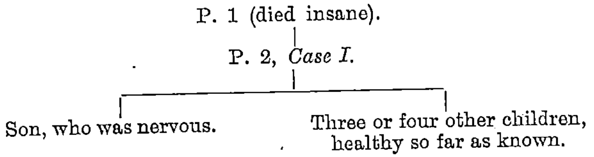
Reaction of tongue and facial muscles to electricity normal.

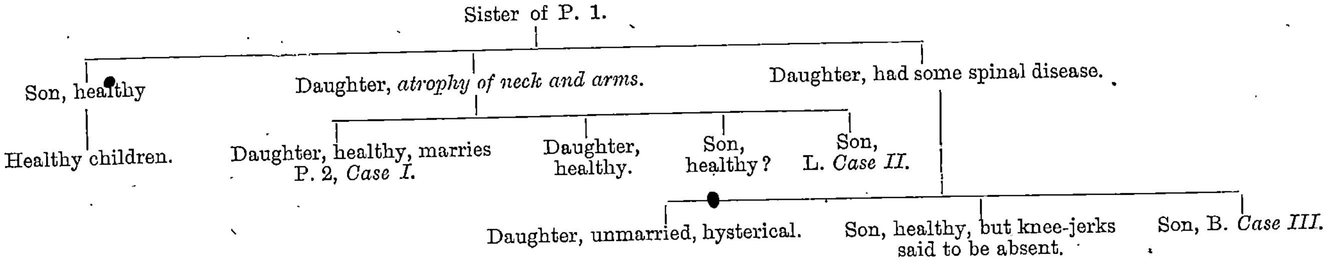
Lower limbs normal.

The patient died three months later.

The mother of Case 2 appears to have had the disease; according to her medical attendant she had wasting and palsy of the muscles of the neck and arm, and had to be fed.

The author evidently considers (though he admits that without a *post-mortem* rigid proof is wanting) that this affection is due to disease of the spinal and bulbar motor cells (myelopathic atrophy). It differs from myopathic atrophy (dystrophia muscularis progressive of Erb) in the early affection of the deep


P. 1 (died insane).
P. 2, *Case I.*
Son, who was nervous.
Three or four other children, healthy so far as known.


Sister of P. 1.
Son, healthy
Healthy children.
Daughter, *atrophy of neck and arms.*
Daughter, healthy, marries P. 2, *Case I.*
Daughter, healthy.
Son, healthy?
Son, L. *Case II.*
Daughter, had some spinal disease.
Daughter, unmarried, hysterical.
Son, healthy, but knee-jerks said to be absent.
Son, B. *Case III.*

muscles of the neck, in the speedy spread to the bulbar nuclei, and the (consequently) rapidly fatal course.

From the known types of myelopathic atrophy, however, it differs in the prominent part which heredity appears to take in its production, for it is generally held that progressive muscular atrophy of spinal origin does not run in families.

The hereditary nature of the affection is best illustrated by quoting the family tree given by the author.

From this it appears that nothing is known of the originators of this diseased stock (father and mother of P. 1); that in the second generation from them the disease appeared in two or perhaps three cousins; and, in the third generation, again in two cousins; lastly, it may be noticed that P. 2, a victim of the disease, intermarries with the daughter of his (diseased) cousin. Considerable interest, therefore, would attach to the children of this couple, but so far, at least, the disease had not appeared in them. P. 2 (Case 1) must have obtained the liability to the disease through his father, the others had it through descent in the female line.

Groenow on Double-sided Hemianopsia of Central Origin. (*Archiv f. Pysch.* xxiii., p. 339.) The first case of this kind has been described by Foerster. The author records a second, and refers also to a patient of Charcot's, who presented a similar loss of sense of locality. He argues that here also a double hemianopsia must have existed.[1]

Concentric narrowing of the half fields of vision in lateral hemianopsia is, as a rule, not to be regarded as depending on a primary disease of both visual centres. Hemianopsia, which may be referred to double-sided brain disease, is more probably indicated when the boundary between the defective and sound parts of the visual field considerably encroaches in places towards the active side (*e.g.*, as when beyond total loss of left half fields of vision of both eyes a partial defect occurs in the right). For a case to be absolutely typical, it is requisite that the halves of the field of vision supplied by one optic tract should be impaired in a first attack, and the halves supplied by the other tract in a second. These considerations are fulfilled by the following case:—

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[1] Charcot's case will be found described on p. 152, *et seq.*, of N.S.S. translation of Charcot's Works, vol. iii.

A sculptor, Professor X., aged 58, had on 10th January, 1889, an attack of left-sided hemiplegia; the arm and leg were slightly paralysed, but he was able to use them. There was no loss of consciousness, but he noticed on resuming work a disturbance of sight, for which at the end of April, 1889, he consulted a doctor. Complete left-sided hemianopsia was diagnosed, with normal sharpness of vision. Ten months later he had another apoplectiform seizure, when he became forgetful, languid and sleepy. He slept for nearly two days, but could be roused without difficulty for his meals. Three days after this last attack it was found that he had lost the power of finding his way about; he believed himself to be in a strange dwelling, and demanded to be taken home; otherwise he seemed clear in his mind. His sight was disturbed, and though there was no absolute amaurosis, yet he could not, *e.g.* recognise some men at work opposite his house. His field of vision was found to be extraordinarily restricted. Sharpness of vision in certain positions of head was good. He gradually improved so that he could find his way about his room alone. December 15th, Dr. Groenow first saw him. Smell, taste and hearing unaffected by attacks. Sense of position of limbs in space normal. Slight impairment of strength in left hand, sensibility to pricking normal. Sharpness of sight good; movements of eyeball free; no abnormal ophthalmoscopic appearances. The field of vision of each eye, however, is reduced to an irregular figure of 8, obliquely placed; the right lower and left upper quadrants in each eye being defective, though not absolutely blind. Coloured objects 5 mm. square were correctly recognised only in a limited central region scarcely reaching two degrees from the perpendicular through fixation point to the left, but nearly ten to the right upper. It was slightly narrower for green than red and blue. The left halves of field of vision, which were lost at first attack, still very imperfect; colour vision is completely absent, and he can only distinguish between light and darkness. In November, 1890, a portion of the inner lower quadrant of the right eye had recovered, and two (possibly overlooked) defects to the left of fixation point were discovered. The intactness of vision in the neighbourhood of the fixation point, in this case as in others, supports Foerster's hypothesis that the frequency of the deviation of the separation line towards the defective side is not due to a blending of the fibres of both optic tracts in each retina, but to a more favourable blood supply in the centres of occipital lobe which subserve the function of sharpest vision.

Colour-sense normal in the restricted field. The author remarks that this fact, compared with the absolute colour-blindness in Foerster's case, is perhaps due to the greater extent of the field in X.'s case, and confirms Foerster's contention that for the distinction of colour a large cortical surface is required. The latter must have been more impaired in Foerster's case. Colour-memory good; no phosphenes, but he experiences "round the eye a wave-like motion like water rippled by the wind." No aphasia; reads and writes well, both from memory and dictation.

Almost complete loss of the sense of locality. Everything since the second attack makes a strange impression on the patient; he does not recognise his own room, nor can he find his way about his house. If placed, however, in the corner of a room, and a door is pointed out to him, he goes directly to it (even with closed eyes), avoiding any obstacles. His power of orientation in the open is very defective. All streets appear much longer than formerly, but not so periods of time. Judging distances at first difficult, all objects appeared to be nearer than in reality, but soon corrected. His mental condition otherwise normal, except slight forgetfulness; quite conscious of his deficiencies. The different psychical functions related to the sight-sense also found fairly normal, except a considerable deficiency in the number of his visual memories. Older impressions better preserved. Fresh visual memories retained for some minutes only. Assisted himself by touch impressions. His geographical memory is practically gone.

In discussing the symptoms of this case, Dr. Groenow comes to the conclusion that the lost power of orientation and memory of locality is to be referred to this partial loss of visual memories. It is possible to acquire correct topographical impressions through the senses of touch and space, as well as by description, in cases where occipital lesions have destroyed the visual locality sense. A man, blind from peripheral lesion, could after a very few days find his way about a room guided solely by the sense of touch; this X. could not do. The presumption is that every representation of space acquired through touch excites simultaneously the corresponding visual representation, and that it is this secondarily excited visual picture which is mainly preserved.

The latest accounts of the patient show that he improved considerably while taking iodide of potassium, he gradually learnt to orientate himself better, but there was no alteration in the limits of the field of vision.

The conclusions that Dr. Groenow draws from this and the other cases referred to are:—

I. If in cases of bilateral hemianopsia of central origin one part of the field of vision remains intact, that this part contains as a rule (perhaps always) the fixation point.

II. The disturbances of sense of locality occuring in lesions of the occipital lobe rest upon the loss of numerous visual memories.

J. Turner.

Mahaim on the Fillet. (*Archiv für Psychiatrie*, vol. xxv. pp. 343-382.)

In a very exhaustive and fully illustrated account of a case of limited lesion on the right side of the cerebrum, the author draws conclusions with regard to the connections of the fillet which are at variance with those expressed by Flechsig, Hösel and others. According to him, the fibres of the fillet are not directly connected with the cerebral cortex, but terminate above, for the most part at least, in the optic thalamus.

The lesion, which was apparently an old softening, was limited to the supra-marginal and superior temporal convolutions, and to the white matter underlying the central convolutions; to the external division of the lenticular nucleus and the whole of the internal capsule, except a small portion posteriorly and the optic radiation from corpus geniculatum externum to the occipital lobe. In the optic thalamus the external nucleus was entirely absent, the internal nucleus showed a great diminution in its cells, and, while the internal medullary lamina was absent, the external medullary lamina and the lattice layer were present, but smaller than on the left.

Mahaim found that there was only partial atrophy of the median fillet of the same side, the inter-olivary stratum and the internal arcuate fibres of the opposite side, and not such an extensive disappearance of those fibres as should have been present if the view that the motor cortex is their nutritive centre is correct. From this Mahaim infers that the atrophy of the fillet was merely secondary to the disappearance of the cells in the optic thalamus, and, except to a limited extent, could not have resulted from the cortical lesion directly. The results obtained should be compared with those described by Hösel (in vol. xxiv. of the same *Archiv*), in which a somewhat more extensive lesion of the central convolutions was followed by a complete degeneration of the median fillet, although the optic thalamus had remained intact.

It seems difficult to reconcile those opposing views if one can be satisfied that the method of staining adopted by Mahaim is sufficient to demonstrate completely that the internal capsule and cortex in his case, to which the fillet fibres are supposed to pass by Hösel, were completely destroyed. The writer of this abstract publishes a case of complete descending degeneration in the median fillet as a result of a lesion in the optic thalamus, the internal capsule and the lenticular nucleus, and he has under examination at present a case of extensive destruction of the cortex of the occipital lobe and post-central convolutions, in which the condition of the fillet appears closely analogous to that of Mahaim's case. These two cases would appear rather to support the view that the fillet terminates in one or other of the basal ganglia rather than in the cerebral cortex. It seems probable, from other considerations, that there is a trophic centre for the median fillet in the nuclei of the posterior columns as well as in its upper termination, and that, under certain conditions, yet to be determined, but which appear to have some relation to the youth of the individual, the former may long maintain its activity.[1]

ALEXANDER BRUCE, M.D.

Hammond on Progressive Muscular Atrophy.

DR. G. M. HAMMOND exhibited before the American Neurological Association, July, 1893, microscopical specimens, and gave the pathological report of two cases of progressive muscular atrophy, and referred to the fact that considerable confusion is occasioned by the misapplication of the term "peroneal type" to a disease totally dissimilar to the one under consideration.

1.—The first patient was a woman, forty-six years of age, without history of gout, rheumatism, or syphilis. In the fall of 1889, she stumbled over a chair, bruising the right shin slightly, and a few days afterwards she was unable to walk as well as formerly. Soon after, there was marked weakness of the flexors of the foot and the extensors of the toes.

By October, 1890, there was complete paralysis of the entire right side, with reaction of degeneration, loss of power and electrical contractility of the anterior tibial muscles of the left leg, and atrophy and loss of power of the thenar and hypothenar muscles of the right hand.

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[1] (See criticism of Mahaim's views by Hösel: "In Sachen Rindenschleife" Neurol. Ctrblt. No. 17, 1893).

Gradually, but rapidly, the other extremities became involved, and ultimately the abdominal and intercostal muscles were affected. She died suddenly from either cardiac or respiratory paralysis.

The sections of the cord showed sclerosis of the pyramidal tracts and atrophy of cells in the anterior horns, and degeneration of Gowers' column throughout its entire extent. The conclusion to be drawn from a clinico-pathological study of this case would be that the muscles of the hand were supplied by the mesial group, and the muscles of the forearm from the anterior group of cells in the anterior horn.

2.—In the second case, first seen in 1882, the disease began in the muscles of the left thumb. He had the opportunity to watch the case to its termination, in 1893. It ran the usual course, and was in every way typical. At the time of death all of the muscles from the level of the armpits upwards, except the facial muscles, were atrophied, as well as those of the upper extremities. She became maniacal, and finally died from exhaustion.

The cord and medulla were examined, and the changes were identical with those described in the preceding case.

He concluded that these cases demonstrate that progressive muscular atrophy is due to a degeneration of the cells in the anterior gray masses and the nerve tubes in the antero-lateral white columns. Also that it is superfluous to divide progressive muscular atrophy into different types because the disease happens to begin in different groups of muscles. (*New York Medical Journal*, January 6th, 1894).

Laycock on a Case of Brain Injury, with loss of Muscular Sense.

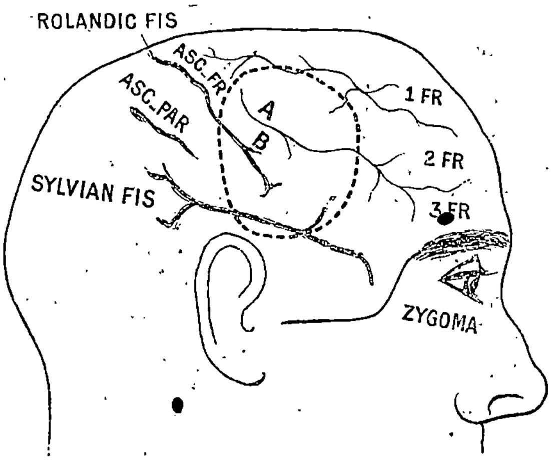
On 24th February last, while the patient was cutting down a tall gum tree, a branch about the thickness of a man's arm fell from the height of about 100 feet on his head. The patient was standing firmly in position for swinging his axe, and the falling branch meeting what was practically a rigid body, simply crushed in the skull, just behind the right parieto-frontal suture. The patient was admitted to the hospital next day. He was then unconscious, with great restlessness and delirium. The right eye was contused, the pupils were equal, and there were left hemiplegic symptoms. The vertex was extensively fractured, and in the wound there were pieces of bone and brain substance. Mr. Cooke cut down over the wound and removed ten pieces of bone, the gap in the cranium being an irregular oval measuring four by three inches. He explored the wound with his finger, and found the base of the skull fractured just behind the orbits, there being a fissure about a quarter of an inch wide. The right frontal bone could be easily moved, and in fact the skull had been practically split in two in the line of the blow. A good deal of lacerated and contused brain substance was removed, the edges of the dura mater brought together as far as possible, and the wound closed.

After the operation, the patient was very restless, extremely noisy in his delirium, and was continually throwing his right arm about. The nurse reported that he moved his arm in a circular direction, and that if anybody was speaking within earshot of him, he would catch up the last two or three words and repeat them several times. The hemiplegic symptoms were well marked. There was scarcely any rise of temperature.

On the sixth day after the accident, consciousness began to return, and he could recognise people about him. At this time there was noticed right facial paralysis; this, however, passed off in about four weeks. From this time he gradually became more rational, though very restless and excitable. On the tenth day a hernia cerebri began to threaten, but this soon passed off. The improvement continued, and in about a month the hemiplegic symptoms began to disappear.

During his illness he complained from time to time of a cramping sensation in the flexors of the right forearm and a slight pain in the part, but there was no rigidity or tenderness of any kind. He continued very emotional, but in every other respect the progress was continuous, and on the 4th of May, just ten weeks after the accident, he got up.

Laycock first saw him at this stage, and his condition was as follows:—He could walk with assistance, but in a very uncertain manner, his gait being distinctly hemiplegic. He had good average power of grasp in the left hand, and the muscular power in the left leg seemed unimpaired. Voluntary movements were performed with the left limbs in an extremely awkward manner. He had complete loss of sense of position in the leg, and incomplete in the arm. If his leg were moved when his eyes were shut, he could not tell where it had been moved to. Under similar circumstances he could tell the position of his arm, but with considerable uncertainty. He could recall in idea a series of positions, the change being made with his eyes shut. He could move the muscles of his face, but could not make a specified movement. He said he could do it, but did not know how. The movements of the eyeball and eyelids were perfect, also those concerned in speech. The sense of weight was not tested, but the allied sense of resistance was almost absent. When his hand was pressed down, say on the bed, and he was told to move it towards his face, his eyes being shut, he was incapable of bringing the requisite amount of force in the proper direction, and made no difference in the amount of force he used on the pressure being increased or decreased; also he was quite unable to disengage his hand from one pressing it down, and struggled in an aimless kind of manner. On the right side there was no symptom, except a crampy feeling in the right forearm. There was, however, no spasm or motor disturbance, and no loss of strength at that time. The right leg was quite unaffected. The knee-jerks and plantar reflexes were absent on both sides



The dotted line shows the extent of the injury to the bone, the letters A and B are placed over the deepest part.

As regards sensation: On the affected side he had sensation of a very rudimentary description, there being only tactile and painful sensibility. When he was touched he felt he was touched, but did not know what kind of thing had touched, or where the point was in the limb. He stated that soon after he regained consciousness he felt a fly walking over the back of his hand, but he did not know that it was a fly, or where it was without looking for it. If his arm were touched, his eyes being shut, he marked the spot two or three inches below the point. If his arm were slightly pinched, say on the outside of the elbow, he felt the pinch in the fingers, or as he described it, between the ring and forefingers. If on the inner side of the elbow, the sensation was between the little and ring fingers. If the arm were strongly pinched the pain shot all over it, without being localised in any way. In the skin of the face there seemed to be a delay between the contact and the mental conception. The same result could be got from the arms, but not so marked. He could not distinguish heat from cold, but if the heat of the water were excessive, pain was caused which spread all over the arm. In the leg, the sensory disturbances seemed to run in the same lines, but the symptoms could not be so clearly brought out.

The depression on the head forms an irregularly-shaped oval, 9 cm. by 7 cm., its deepest points A and B in the diagram being 3.7 cm. and 3.5 cm. respectively, measured from the perpendicular plane to the external meatus. The posterior wall slopes abruptly to the deepest part, the anterior more gradually. The upper margin is 5.5 cm. from the middle line at a point 17 cm. from the nasion, and the lower margin is a similar distance from the zygomatic line. (For purposes of comparison, the dimensions of the patient's head are given in the middle line from the root of the nose to the occipital protuberance 36.2 cm. Laterally, between these two points, 28.75 cm. Between the external meatus 15.5 cm., from the external meatus to the middle line at the vertex 18.5 cm., cutting the middle line 17.5 cm. and the lateral line at 14.7 cm. from the nasion.)

The following parts of the cerebrum would appear to be involved:—The lower half of the ascending frontal convolution, and the greater part of the sigmoid flexure, the posterior third of the lower and middle frontal convolutions, and the base of the posterior end of the upper one; also the base of the corresponding part of the falciform lobe. The destruction being deep at the part the nerve fibres radiate from the anterior half of the internal capsule, these fibres must have been affected close to their emergence. Thus it will be seen that the motor centre for the arm would be the one principally involved. Those of the leg and trunk and of the face would be affected to a less degree.

As regards his other senses—hearing, taste, and smell are quite unaffected. Speech also, and the promptitude with which he answers questions, forms a marked contrast to the slow and apparently stupid manner in which he performs movements with his affected arm.

Ophthalmological examination.—Fundus rather pale; veins enlarged; arteries small; muscles of the eye, both external and internal, normal; vision, as far as it was tested, was quite normal.

His temper seemed quite unaffected, and his memory and intelligence were perfect. He is rather emotional, and inclined to laugh or cry. The rest of the body is sound and healthy, without the slightest indication of any disease.

As soon as the wound had completely healed up, and the patient was able to bear it, L. endeavoured to get some localising indications from electrical stimulus. The indifferent pole, a flat-electrode, 5 by 8 cm., was placed on the back of the neck, the other pole, with a small olive-shaped point, was placed on various parts of the depression. No result was obtained from the faradic current, the patient being able to bear only a very mild degree of strength. A galvanic current of 5 to 10 milliamperes yielded the following results :—With the anode in the inferior part of the posterior margin—the face centre—on each interruption of the current there was a sensation in the neck on the opposite side. With the kathode, the sensation was more localised, and went through both sides of the neck just above the level of the angle of the jaw. With the anode in the middle of the posterior margin—the arm centre—there was a sensation in the shoulder and upper part of the arm. With the kathode, the sensation extended down the arm in a slow crawling manner (to use the patient's expression), and when it got to the fingers there was a tingling as if the hand had been numb with cold, and had been brought to a fire. The patient was able to follow the sensation downward with his finger. On the current being increased to 12 or 13 milliamperes, both sides were affected; the right, however, not so much as the left, the sensation downwards being of much greater rapidity. On the anode being placed on the upper part of the posterior margin—the centre for the trunk and thigh—there was a sensation just at the angle of the neck and shoulder. With the kathode the sensation was in the same region, but somewhat deeper from the surface. In none of these observations was any muscular action obtained. The sensations were not painful, being only of a tingling character, and it should be noted that the upward current gave far more definite results than the downward.

As regards the subsequent history, the patient has improved steadily in the condition of his left arm and foot, and performs movements with them much more expertly. The right arm, however, is showing signs of weakness. When L. last saw him, the crampy sensation in the flexors of the hand and wrist was still there, and it was accompanied by a burning pain at the same region. There was no tenderness or swelling, and no headache of any kind. The pain was best relieved by simple rubbing.

On reviewing the history, there are one or two points of interest. From the nature of the injury, a zone of brain substance just in front of the fissure of Rolando would bear the brunt of the blow, and its left half would, at least, be severely concussed. This left half would include Broca's convolution (the centre for speech), and the anterior end of the temporal lobe (one of the auditory centres). It will be remembered that it was noted that, for two or three days after the accident, the patient was very noisy, and if anyone were talking in his presence, he would catch up the last two or three words of the sentence and repeat them several times, he being unconscious at the time. This would signify that the auditory and speech centres were in a state of extreme excitability, and that the stimulation by the vibration of the sound of a word would set automatically into action the whole chain of centres, the result being the spoken words. The zone of brain substance would also include the centre for the right arm. During the first two or three days he threw about this arm a great deal, chiefly in a revolving manner, which showed its cortical centre to be in a highly irritable state. The symptoms of cramp and burning pain, and the weakness which has come on more recently, have certainly a causal connection with this, and it seems to me to be rather ominous, and shows that there is some mischief, probably of an inflammatory character, going on in the left frontal lobe.

The cerebral injury presents a problem of the greatest complexity. With the exception of a small part of the base of the falciform lobe, which is considered to be sensory, the bulk of the part destroyed is in the area which is generally regarded as motor. Nevertheless, the sensory and motor functions of, say, the arm, as it is the part most completely involved, seem to be affected in a relatively equal degree, and both of these functions seem to have been cut down to a very rudimentary level.

On the sensory, or to be more exact, the afferent side, there is simple tactile sensibility, but there is an absence or deficiency of the higher conceptions of sensation, which are evolved from the lower and simpler levels, such as the position of the limb, the qualities and position of foreign bodies touching the part, their weight, resistance, &c. Similarly on the motor or efferent side, there is the power of making simple movements, but there is an absence or deficiency of the power of making the more complicated ones, which must necessarily be on a higher developmental level.

As both these afferent and efferent functional disturbances are the result of one and the same injury, it follows that the part destroyed, that is, part of the Rolandic area, is sensori-motor. What, however, the comparative importance of each of these factors is, and where their functions end and begin, are the most difficult problems in neurology. These afferent impressions make up the complex which forms the so-called muscular sense, and as they are severely affected, especially in the arm in the case under discussion, the symptoms of loss of this sense are very well seen. In the group of impressions and sensations which form this sense, L. is inclined to regard the sense of position as of the greatest importance, and the awkwardness of movement in this case depends more upon this than upon anything else. The nicety of muscular movement, and the quantity of force used, entirely depend on a knowledge of the point from which the muscular force has to commence action, and of the successive changes of position or relations which the movement causes.

These estimations of position are always present in the normal body, as anyone can ascertain for himself, and it leads me to the belief that they are not of the nature of ordinary sensory impressions, such as pain or heat and cold, but that there must be some arrangement by which a circuit of nerve force is continually in equilibrium. In the cerebral part of this circuit there will be some sort of centre like a galvanometer in an electric circuit, which will keep the cortical centres informed, so to speak, of the state of the peripheral parts, not only when they are at rest, but when they are in action, action being indicated by change from one state of equilibrium to that of another. In our case, injury to the cortical loop of such a circuit could explain many of the symptoms.

As regards the sensory disturbance, here again the sense of position takes an important place. Common or cutaneous sensation, with pain or some variety of pain, has its nervous tract in the posterior end of the hindmost limb of the internal capsule. This has escaped injury in our case evidently, for common sensation seems to be actually increased. The sense of tactile localisation is almost lost, and L. believes we may infer that this faculty is in a different nervous tract from common sensation. In the diagrams of the internal capsule, the anterior half of the anterior limb is left blank. From the experience gained by this case, L. thinks it may be fairly filled up by the tracts of the so-called muscular sense, or, as L. prefers to call it, the sense of local relation.—(Abridged from the *Australian Medical Journal*, July, 1893.)